THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—19TH YEAR. SYDNEY, SATURDAY, SEPTEMBER 10, 1932. No. 11.

Table of Contents

THE INCIDENCE OF PUNCTATE BASOPHILIA.

By W. T. NELSON, M.D., M.R.C.P.,
Commonwealth Serum Laboratories,

LIONEL LOCKWOOD, M.D.,
Surgeon Lieutenant Commander, Royal Australian Navy,

AND

KATE MACKAY, M.D.,
Department of Labour, Victoria.

(From the Commonwealth Serum Laboratories, Melbourne.)

THE work described in this paper arose out of a previous inquiry[1] into blood changes in workers in trinitrotoluene and in two other classes of workers. By the use of a dark field technique for examining films, unexpectedly large numbers of stippled cells were found; but, as the men in the control groups previously examined were comparatively few and were not proper subjects for strictly normal controls, it was decided to examine, by the new technique, blood films from naval ratings and trainees who were undoubtedly representative of healthy types of young adult workers. In addition, blood films were examined from workers in lead battery factories.

The Findings of Other Workers.

Various investigators of punctate basophilia in normal subjects have reported findings very different from ours, but they employed a different technique. Brown[2] reports that in blood smears from fifty-five apparently normal persons, "no evidence of stippled cells in any specimen was found". Mayers,[3] in an examination of blood films from 250 normal persons, found one stippled cell. Kogan and Smirnowa[4] state:

> In men not working in lead industries, there was not one instance of basophilic granules without anæmia.

On the other hand, according to Price-Jones,[5] it is well established that cells showing punctate

basophilia may occur in quite healthy persons, and that they are observed in great numbers in various diseased conditions associated with anæmia.

McDonald[6] quotes Duhig as having found punctate basophilia in 43% of one hundred children, and states:

> The bulk of these children were entirely free from any manifestation which could be considered, however remotely, as due to lead absorption.

Aub, Fairhall, Minot, and Reznikoff[7] write: "Even normal blood contains a very few stippled cells." Lane[8] found punctate basophilia in about half of 223 normal controls. He also pointed out the desirability of a quantitative estimation of this condition.

In regard to the question whether punctate basophilia be found in the bone marrow, Aub, Fairhall, Minot, and Reznikoff[7] cite seven references to literature in support of their assertion that it is absent. However, Ehrlich and Lazarus[9] quote four authorities stating that it is present in the bone marrow, and in this opinion they are supported by Price-Jones[5] and by Drinker.[10]

The Demonstration of Punctate Basophilia.

The demonstration of punctate basophilia, when present in blood films, may be easy or difficult according to the technique used. It is necessary, as pointed out before,[1] to examine the films in a fresh state; to use a stain which is most likely to show stippling, such as an alkaline methylene-blue solution;[7][11] and, finally, in searching films, to employ a dark field technique enabling one to find not only very coarsely stippled cells, but also cells with finer granules, many of which are not apparent in light-ground illumination. When the technique is adequate, stippling of red cells is commonly found, not only in persons with pathological activity of the red cell regenerative centres, as in lead poisoning and pernicious anæmia, but also in healthy young adult workers.

The Present Investigation.

The original purpose of this investigation was to make accurate counts of the various types of stippled cells in a large number of normal young adult workers and then to compare the results with those obtained from men suffering from lead poisoning or exposed to that occupational risk. In this connexion Badham and Taylor[12] state:

> Quite apart from the scientific interest which attaches to the interesting pathological changes that lead poisoning produces in humans, from a medico-legal standpoint there are many problems associated with it, and these chiefly centre round the standards which may properly be employed to diagnose lead poisoning.

Blood films from 314 naval ratings and trainees were examined. These were mostly young male adults, only 6·5% being over thirty years of age. Of these, 168 were naval ratings and 146 were naval trainees, young men drawn from ordinary civil life for a course of naval training. In each case a careful history was taken of the possibility of any hazard that might of itself be accepted as a causal agent of punctate basophilia, particularly lead poisoning. Similarly, blood films were obtained from 41 workers in factories manufacturing lead storage batteries. An attempt was made to establish the clinical diagnosis of the presence or absence of lead poisoning in these cases. Blood films were fixed with methyl alcohol and then forwarded to the laboratory. They were there stained by Sellers's method[11] and examined by the dark field technique as described,[1] with the exception that a "net" micrometer in one of the oculars was included to facilitate counting. The results of the stippled cell counts of the various classes are shown in Tables I and II.

TABLE I.

Type of Subject.	Average Number per Man of Stippled Cells per Million Red Cells.
255 men with no ascertained lead hazard	937
59 men with a history of exposure to a slight lead hazard	4,335
41 workers in lead battery factories	14,220
12 workers from lead battery factories suffering from lead poisoning	36,382

These results show that the average total stippled cell count of the normal controls with no history

TABLE II.

Subjects.	Men with No Ascertained Lead Hazard, 255.		Men with a History of Slight Exposure to Lead, 59.		Workers in Lead Battery Factories, 41.	
	Number.	Percentage.	Number.	Percentage.	Number.	Percentage.
Those in whose films no stippled cells were seen	80	31·3	6	10·2	2	4·9
Those with 110-1,000 stippled cells per million red cells	107	42·0	5	8·5	4	9·8
Those with 1,001-2,000 stippled cells per million red cells	32	12·6	7	11·8	3	7·3
Those with 2,001-5,000 stippled cells per million red cells	27	10·6	22	37·3	8	19·5
Those with 5,001-10,000 stippled cells per million red cells	9	3·5	13	22·0	8	19·5
Those with 10,001-15,000 stippled cells per million red cells			5	8·5	3	7·3
Those with 15,001-20,000 stippled cells per million red cells			1	1·7	1	2·4
Those with more than 20,000 stippled cells per million red cells					12	29·3[1]

[1] These were all suffering from lead poisoning.

of exposure to a lead hazard is under 1,000 per million. None of these men has a count exceeding 10,000. All men suffering from well marked lead poisoning have more than 20,000 punctate cells. No endeavour was made to correlate stippled cell counts with degrees of plumbism.

In view of the relatively high percentage of carbon monoxide in tobacco smoke reported by Willcox,[13] it was decided to ascertain whether smokers as a class had higher stippled cell counts than non-smokers. The results are shown in Table III.

TABLE III.

(a) *Smokers with No Lead Hazard*, 199.

Subjects.	Number.	Percentage.
Those with no stippled cells ..	66	33·2
Those with 110-1,000 stippled cells per million red cells .	83	41·7
Those with 1,001-2,000 stippled cells per million red cells ..	22	11·1
Those with 2,001-5,000 stippled cells per million red cells ..	21	10·5
Those with more than 5,000 stippled cells per million red cells	7	3·5

(b) *Non-Smokers with No Lead Hazard*, 56.

Those with no stippled cells ..	14	25·0
Those with 110-1,000 stippled cells per million red cells ..	24	42·9
Those with 1,001-2,000 stippled cells per million red cells ..	10	17·8
Those with 2,001-5,000 stippled cells per million red cells ..	6	10·7
Those with more than 5,000 stippled cells per million red cells	2	3·6

The results shown in Table III fail to show any difference between smokers and non-smokers in their stippled cell counts. In this connexion Ramsey and Eilmann[14] state: "Gruber has shown that CO is not a cumulative poison, when inhaled in very small amounts it rapidly disappears from the blood. Therefore, the blood examination may not be an aid to diagnosis in chronic cases" of carbon monoxide poisoning.

TABLE IV.

Particulars of Nine Men with No Lead Hazard, but with Stippled Cell Counts Exceeding 5,000 per Million Red Cells.

Occupation.	Age, in Years.	State of Health.	Stippled Cell Count.
Steward	25	Normal	5,170
Steward	27	Normal	9,900
Dentist	28	Normal	8,910
Seaman	28	Normal	8,910
Labourer	28	Normal	5,500
Labourer	22	Normal	7,260
Clerk	19	Normal	7,260
Wool-classer	19	Normal	5,280
Labourer	22	Normal	5,610

In Table V the particular lead hazard of each man with a history of contact with lead is briefly indicated and the corresponding stippled cell count is given.

Attention is drawn to certain comparatively high stippled cell counts accompanied by apparently slight exposure to lead. Likewise one observes that high counts are present for a considerable length of time following the operation of painting ship. "Grey" paint, which is used most, contains about 19% by weight of white lead; "white" paint contains about 25%, while "red lead" paint has about 75% of red lead and white lead in its composition.

TABLE V.

Subject and Hazard.	Number of Stippled Cells per Million Red Cells.
Stoker, coppersmith's assistant: Involves working with lead	2,750
Seaman: Painted ship nine months ago; no contact with lead since	15,000
Said to have had lead poisoning three years ago	0
Painted ship eighteen months ago	3,100
Electrician—torpedoes: Handles quantities of lead-enclosed wires	2,600
Seaman: Maintenance and repair of storage batteries	10,450
Seaman: Does a little painting	1,100
Coppersmith's assistant: Works with lead; painted ship two years ago	2,750
Painted ship twelve months ago	4,600
Painted ship eighteen months ago	9,900
Painted ship eighteen months ago	4,950
Painted ship three months ago	5,280
Worked for three years with storage batteries until eighteen months ago	4,620
Worked in double bottoms, which are painted with red lead	3,300
Painted ship twelve months ago and for three years prior to that	2,860
Has been painting ship recently	2,970
Painted ship twelve months ago and for six years previously	2,310
States he had lead poisoning eighteen months ago from work in submarines	4,840
Painted ship four months ago and for three years prior to that	5,060
Painted ship three months ago	4,620
Painted ship seven months ago and twice yearly for five years prior to that	15,840
Painted ship three months ago	9,900
Continually working with metals, including lead ..	6,270
Has been working with storage batteries for three weeks	10,890
Prior to six months ago was at sea for four years, during which time he continually painted ship ..	8,240
Continually working with metals, including lead ..	1,320
Painted ship twelve months ago	2,970
Painted ship six months ago	7,260
Metal turner	5,710
Printer: Works with lead type	1,100
Metal turner	5,940
Metal turner	4,950
Painted boats for two weeks two years ago	8,580
Printer: Works with lead type	3,300
Printer: Works with lead type	1,980
Carpenter: Contact with paint	1,540
Small amount of painting six months ago	3,740
Tiler and slater: Works occasionally with lead and paint	1,540
Seaman: Uses lead paint four days per week	4,620
Plumber: Has used lead occasionally, but not for the last twelve months	5,060
Bricklayer: Uses lead paint occasionally	10,340
Orchardist: Sprays trees	10,560
Engineer: Lead rarely	2,420
Plumber: Has slight contact with lead	4,180
Fitter and turner (metal worker)	6,600
Plumber	0
Plumber: Has slight contact with lead	1,980
Linotype mechanic: Slight contact with lead	8,800
Artist	220
Jeweller: Slight contact with lead	2,200
Electrician: Slight contact with lead	3,850
Electrician's mechanic: Works with accumulators	0
Printer: Works with lead type	0
Ironworker, with slight lead risk	0
Enameller	0
Engineer: Works with lead piping	660
Engineer: Works with lead piping and paint	660
Handles duco paint and batteries	770
Ironfounder: Uses lead occasionally	660

Workers in Lead Battery Factories.

For purposes of comparison, blood films from forty-one workers in lead battery factories in Melbourne were obtained. In Table VI are shown the stippled cell counts of these, together with the

results of the clinical examination as to the presence or absence of lead poisoning.

TABLE VI.

Occupation.	Duration.	Clinical Diagnosis.	Stippled Cells.
Mixer	2 years		1,760
Paster	3½ years		15,290
Moulder	3 years		0
Moulder	3 weeks		2,970
Charging and occasionally mixing	—		7,260
Lead burning	12 months	Lead poisoning	33,000
Lead burning	3 years		4,620
Moulding and burning	3-4 years		12,870
Clerical work	18 months		4,180
All processes	Many years		990
Moulding	2 years		2,200
Moulding	3 years		2,640
Burning	2 years		12,540
Pasting	1½ years		5,280
Charging	3 years		110
Putting plates in tanks	5 months		3,300
Lead burning	1½ years		0
Foreman	9-10 years		6,380
Mixing	3 years	Lead poisoning	57,420
Burning, pasting	4 years		1,650
Mixing, forming	8 years		8,360
Moulding	4 years		2,640
Paster	1½ years	Lead poisoning	44,880
Repairing	6 years		1,650
Stamping containers	1½ years		9,240
Packing *et cetera*	2 months		550
Paster	3 years	Lead poisoning	46,200
Moulder	2 months		7,590
Manager—all jobs	Years		770
Moulding, pasting	—		4,070
Clerical work	2 years		8,360
Lead burner	—	Lead poisoning	43,560
Mixing and pasting	2 months		7,260
Mixing and pasting	2 months	Lead poisoning	34,100
Smelting	3-4 years	Lead poisoning	33,660
Burning and charging	2 months		11,880
Smelting	2 years	Lead poisoning	23,650
Paster	6 weeks	Lead poisoning	26,400
Paster	12 months	Lead poisoning	31,020
Paster	8 weeks	Lead poisoning	36,300
Paster	2 months	Lead poisoning	26,400

In 199 of the examinees the stippled cells were classified according to the size of the granules as seen under the dark field microscope, under the headings of "fine", "medium" and "coarse". In the usual bright field examination the cells with fine granules do not show the stippling, but the medium and coarse varieties are visible. In Table VII are given the results of the classification of all the stippled cells found in the blood films from these men.

This table shows that the coarsest variety of stippling forms an apparently large proportion, roughly one-seventh, of stippled cells in those persons without a history of contact with lead. In other words, this grade of stippling is not a specific qualitative phenomenon associated with absorption of lead or other poison. On the other hand, the coarsest stippling does appear to bear some quantitative relationship to lead absorption; for, as the degree of the lead hazard increases, the percentage of this variety of stippling increases. In lead poisoning it is twice that found in those without a lead hazard.

In a hypothetical case of a lead storage battery factory worker with anæmia and a high stippled cell count, it is as well to bear in mind that he may be suffering, not from plumbism, but from pernicious anæmia or some other disease. In this connexion Price-Jones[5] states:

> They (stippled cells) are observed in great numbers in various diseased conditions associated with anæmia.

Ehrlich and Lazarus[9] state:

> Punctate erythrocytes may be found in all forms of anæmia.
>
> The punctate erythrocytes have been found as the only ascertainable blood change in the stage of complete remission in progressive pernicious anæmia.

In a case of pernicious anæmia referred to us by Dr. Downes, Commonwealth Medical Officer, there were 20,000 stippled cells per million erythrocytes.

The inadequacy of ordinary methods of searching films for stippled or basophilic cells has been recognized by McCord, Minster and Rehm.[15] They write:

> Some means must be found for bringing this substance into greater visual prominence, such as the clumping of this evenly distributed material. . . . The basis of our work is the hæmolysis of the red cell prior to staining.

It is believed by the present writers that the dark field method described,[1] when the Siedentopf change-over condenser is used, fulfils requirements as regards "visual prominence" with no interference with the film. This technique enables one rapidly to make accurate quantitative estimations of all the potentially visible stippled cells in a blood film.

TABLE VII.
Classification of Stippling According to Size of the Granules.

Type of Cell.	Average of 119 Men (Naval Ratings and Trainees) with No History of Exposure to Lead.		Average of 39 Men (Naval Ratings and Trainees) with a History of a Slight Exposure to Lead.		Average of 41 Workers in Battery Factories.		Average of 12 Workers in Battery Factories with Definite Clinical Evidence of Lead Poisoning.	
	Number of Stippled Cells per Million Red Cells.	Percentage of the Average Total.	Number.	Percentage.	Number.	Percentage.	Number.	Percentage.
Cells with "fine" stippling	514	49·5	1,918	45·8	5,650	39·7	13,951	38·3
Cells with "medium" stippling	366	35·2	1,383	33·1	4,516	31·8	11,339	31·2
Cells with "coarse" stippling	159	15·3	878	21·1	4,054	28·5	11,092	30·5

Summary.

1. Estimations of stippled cells were made upon blood films from groups of workers. These were stained by Sellers's method and examined by the dark ground technique.

(*a*) Two hundred and fifty-five men with no history of exposure to lead have an average of 937 stippled cells per million red cells.

(*b*) Fifty-nine men with a history of very slight exposure to lead have 4,335 stippled cells.

(*c*) Forty-one men with a severe lead occupational hazard have 14,220 stippled cells. Twelve men from group (*c*) suffering from lead poisoning have 36,382 stippled cells per million red cells.

2. As the lead hazard becomes greater, the percentage of persons with high stippled cell counts increases.

Of men exposed to no known lead hazard, 3·5% have more than 5,000 stippled cells per million red cells.

Of men exposed to a slight lead hazard, 32·2% have more than 5,000 stippled cells per million red cells.

Of men exposed to a severe lead hazard, 58·5% have more than 5,000 stippled cells per million red cells.

3. Twelve men working in lead accumulator factories and suffering from well marked lead poisoning had each more than 20,000 stippled cells per million erythrocytes.

4. Of 255 men with no history of exposure to lead, none had a count exceeding 10,000 punctate basophilic cells.

5. Amongst the naval ratings and trainees, stippled cell counts exceeding 10,000 were found to have a definite relationship to a lead hazard, even though the exposure were some time previously. Two men of this group with stippled cell counts of 15,000 had painted ship respectively nine months and seven months before this examination was made. There was no history of contact with lead since then. A trainee, an orchardist in civil life and accustomed to spraying trees, had 10,560 stippled cells per million erythrocytes.

6. There appears to be no difference between the appearance of the stippled cells seen in blood films from cases of lead poisoning and those found in normal controls.

7. There is a quantitative difference in regard to the coarsest variety of cell granules, in that, as the degree of the lead hazard of a group of men increases, the proportion of stippled cells exhibiting this grade of punctation increases. In cases of lead poisoning they form 30·5%, while in normal controls they amount to 15·3% of the stippled cells.

8. The persons with lead poisoning had, on an average, thirty-eight times as many stippled cells as the normal controls, and sixty-nine times as many of the coarsely stippled variety.

9. Smokers as a class do not differ from non-smokers in regard to the numbers of stippled cells.

10. The importance of considering all possible causes of high stippled cell counts is illustrated by the fact that a case of pernicious anæmia had 20,000 stippled cells per million erythrocytes.

Acknowledgements.

We desire to thank Dr. F. G. Morgan, Director, Commonwealth Serum Laboratories, for his interest and helpful suggestions; Surgeon Captain L. Darby, R.A.N., Director of Naval Medical Services, for sanctioning this investigation of naval ratings; Surgeon Commander W. J. Carr, R.A.N., Principal Medical Officer, Flinders Naval Hospital, for his cooperation; Mr. C. V. Vaughan, B.C.E., for assistance in examining blood films; Leading Sick Berth Attendants K. Frazer and M. Trulsson for their help in preparing blood films.

References.

(1) W. T. Nelson: "Punctate Basophilia: A Method for Easy Demonstration by Dark Ground Illumination", THE MEDICAL JOURNAL OF AUSTRALIA, March 14, 1931, page 310.
(2) E. W. Brown: "A Study of Lead Poisoning Among Oxy-acetylene Welders in the Scrapping of Naval Vessels", *Journal of Industrial Hygiene*, March, 1926, page 113.
(3) May R. Mayers: "Lead Anæmia", *Journal of Industrial Hygiene*, May, 1926, page 222.
(4) B. Kogan and L. Smirnowa: "Changes in the Blood Picture under the Influence of Lead, and Their Importance in the Differential Diagnosis", *Journal of Industrial Hygiene*, October, 1927, page 435.
(5) C. Price-Jones: "The Effect of Lead on Red Blood Cells", *Medical Science Abstracts and Reviews*, Volume XI, October, 1924-March, 1925, page 147.
(6) S. F. McDonald: "The Minor Manifestations of Plumbism in Childhood", THE MEDICAL JOURNAL OF AUSTRALIA, June 21, 1930, page 806.
(7) Aub, Fairhall, Minot and Reznikoff: "Lead Poisoning", 1926.
(8) R. E. Lane: "The Rôle of Punctate Basophilia in the Control of Industrial Plumbism", *Journal of Industrial Hygiene*, October, 1931, page 276.
(9) Ehrlich and Lazarus: "Anæmia", 1910.
(10) C. K. Drinker: "Oxford Medicine", Volume II, page 518.
(11) A. Sellers: "A Contribution to the Study of Punctate Basophilia in Lead Workers", *Journal of Industrial Hygiene*, April, 1925, page 145.
(12) C. Badham and H. B. Taylor: "Lead Poisoning", Extract from the Report of the Director-General of Public Health, New South Wales, for the year ended December, 1925.
(13) W. Willcox: *The British Medical Journal*, July 5, 1930, page 17.
(14) T. L. Ramsey and H. J. Eilmann: "Carbon Monoxide Acute and Chronic Poisoning and Experimental Studies", *Journal of Laboratory and Clinical Medicine*, Volume XVII, February, 1932, page 415.
(15) C. P. McCord, D. K. Minster and M. Rehm: "The Basophilic Aggregation Test in Lead Poisoning", *The Journal of the American Medical Association*, Volume LXXXII, May 31, 1924, page 1759.

BRIGHT'S DISEASE: ITS CLASSIFICATION.[1]

By G. T. JAMES, B.A., M.D.,
Honorary Physician, Ballarat Hospital, Victoria.

IN the first edition of Dr. Cushney's monograph on the secretion of urine, published in 1917, there appear in a prefatory letter addressed to Professor Starling the following words:

> The growth in the literature on the kidney has been extraordinary and the increase in bulk has not gone along with an improvement of quality, but rather the reverse. No other organ of the body has suffered so much from poor work as the kidney, and in no other region of physiology does so much base coin pass as legal tender.

The growth in the literature did not cease with Cushney's lament. It has gone on increasing year by year, and pathologists, biochemists and physiologists have continued to vie with one another in

[1] Read at a meeting of the Victorian Branch of the British Medical Association, July 23, 1932.

an attempt to explain the vital factors underlying renal failure. The practitioner, who does not deal with the abstract problem of kidneys, but with a human being who possesses kidneys, is justified in choosing from the field of experimental research such material as can be applied.

Therefore, any clinical review of Bright's disease is based on selections that allow a practical classification, a reasonable pathological explanation and as rational a mode of treatment as is possible and consistent with our present incomplete knowledge.

Impairment of Renal Function and Renal Insufficiency.

Reduced to its simplest terms, impairment of renal function means inability of the kidney to concentrate urine to the same degree as a normal kidney would. The discrepancy is compensated, however, by the mechanism of polyuria. This means that a larger amount of urine, but of lower specific gravity, is elaborated, and hence waste products are eliminated in a diluted form. When compensation fails, then renal insufficiency is manifested and retention of waste products commences. The whole conception finds its exact counterpart in cardiac disease, where the day of insufficiency is postponed until compensation fails. A good deal of abuse has been hurled at laboratory methods in renal disease, because they appear to be of use only when compensation fails; but it does not require a laboratory to detect a polyuria with a low concentration of urine, and it must be admitted that too little attention is paid to this important phase of impairment, during which the kidney could be spared a good deal of work by limiting the intake and reducing waste products with which it has to deal.

The modern opinion regarding impairment of function which appears to have received greatest support is that the kidney fails to concentrate a normal urine and fails equally for all urinary constituents. This appears at first sight to be entirely at variance with biochemical findings. For example, in insufficiency the blood urea may be very much increased and the chloride may not. To tell the whole story that this statement suggests would be profitless at this point. Perhaps it would be sufficient to state that the probable explanation of the apparent anomaly is to be found in the way the normal kidney deals with these two substances. For example, the kidney concentrates urea sixty-six times, but chloride only 1·4 times. In other words, urea is concentrated by the kidney about forty-seven times more than is chloride. It is therefore obvious that those substances only slightly concentrated by the kidneys are not demonstrably increased in the blood until the final stages of impairment.

Classification.

A clinical classification may be regarded as a summary of inferences drawn from scientific facts. The number and variety of classifications of Bright's disease that have been evolved from time to time, rather suggests that either no mode of inference is free from the possibility of fallacy or that the scientific statements are not entirely true. For no classification yet enunciated has escaped wreckage in some degree or other in the *post mortem* room. It appears that this subject cannot be divided and subdivided into numbers of artificial and arbitrary pathological entities, because each variety has gradations and overlappings that, in the present state of knowledge, defy pigeon-hole treatment.

There are many schemes that could be considered, but three will afford sufficient material for discussion.

The biochemists, particularly those of the French school, adopted a classification according to what they believed to be the type of impairment present; hence the terms azotæmic and hydræmic nephritis—the nitrogen-retaining and chloride-retaining kidney—corresponding again to the older chronic interstitial and parenchymatous nephritis with œdema. But there is only one type of renal impairment, that in which all the excretory functions are damaged. A biochemical classification would therefore appear both illogical and paradoxical, because it presupposes the very occurrence that biochemistry has proved wrong, namely, selective injury to individual kidney functions.

Secondly, there is Christian's classification, which appears in the "Oxford Medicine". He uses the presence or absence of œdema as a criterion and tabulates a scheme as follows:

Acute nephritis.
Subacute nephritis.
  a. With renal œdema.
  b. Hæmorrhagic nephritis.
Chronic nephritis.
  a. With renal œdema.
  b. Without renal œdema.

Thirdly, there is the modification of Volhard's and Fahr's classification of 1914 and now adopted by Fishberg, Bennett and others. It is as follows:

1. Inflammatory group.
   (*a*) Diffuse.
   (*b*) Focal—Glomerular.
   Interstitial—in acute specific fevers in children.
   Embolic—glomerular.
2. Degenerative group.
   Lipoid nephrosis.
   Chemical nephrosis.
   Amyloid nephrosis.
3. Arteriosclerotic group.
   Essential hypertension.
   Hypertension *plus* renal sclerosis.

From a critical study of these two schemes the present day attitude towards Bright's disease and its classification should emerge. The ideal classification will be written when the ætiology of Bright's disease is no longer a mystery. Failing ætiological factors, a pathological classification is next to be preferred. The use of the terms acute, subacute and chronic is obviously a subterfuge for avoiding a difficult problem, though it may be a convenient subterfuge as a clinical stopgap, simply because it is entirely non-committal as far as the

pathology is concerned. It further destroys, or at least hides, the essential factor for the understanding of glomerulonephritis, namely, its clinical continuity from an initial acute toxic spoiling through its middle stage perplexities of varying cardiovascular renal destruction, to the final phase of renal failure, uræmia and death. The term nephritis for all lesions is inaccurate, even if we omit any discussion on the controversy of what exactly constitutes inflammation and further avoid for the present any reference to the desirability or otherwise of the term nephrosis. The pathological process involved under the heading of chronic nephritis without renal œdema is a widespread one distributed throughout the body, and as far as we know does not even have its beginnings in renal tissue. The arteriosclerotic who has coronary artery degeneration, does not in these days suffer from chronic myocarditis. Why, then, should he be labelled with a chronic nephritis if the manifestation of his arterial degeneration is more obvious in his renal tissue? The next objection is to the use of the presence or absence of œdema as a distinguishing characteristic, particularly when it is qualified as renal œdema. Œdema may occur during the course of any variety of Bright's disease, though the underlying causes for its appearance differ probably in each variety. Even the existence of a true renal œdema is denied by most observers, who postulate a common cause for the renal lesions and extrarenal lesions which cause the œdema.

Of late years the practice has been to accept some such conception of Bright's disease as enunciated by Volhard and Fahr. Using it as a foundation, Miss Russell in 1929 published an intensively elaborated edition of it, which is probably of greater use to the pathologist than to the practitioner. The tendency seems to be even to reduce the original to a simpler form. The main headings are chosen with due regard to the primary histological changes, the secondary changes being regarded as the natural evolution of the disease. The purely inflammatory group is divided from the degenerative group, and for the latter the term nephrosis is reserved. Pure nephrosis is a rare disease and nothing but confusion ensues unless the term is strictly kept for the pure primary tubular degenerations. The object of this remark will be apparent in a moment. Vascular degenerations comprise the third group. Thus a classification of Bright's disease, which conforms with present pathology, and which admits of easy clinical application, recognizes three main classes: (i) the inflammatory or glomerulonephritic group, (ii) the degenerative or nephrotic group, (iii) the arteriosclerotic or nephrosclerotic group.

Before referring to the clinical aspects of these groups, it will be necessary to make some brief reference to the pathogenesis of œdema. The controversies that have raged round the question of the pathology of the nephroses, have at least supplied a voluminous literature on the problems of œdema formation, and many preconceived ideas regarding its formation have now been disproved. No apology is needed for again quoting A. B. Macallum's words written fourteen years ago.

> Behind the functions of the renal organ is a history which links up the human body with the far past, with an age of the earth when its oceans contained what would now be regarded as brackish water, and the earliest type of vertebrate life was just beginning to appear as a marine form. The blood plasma, so far as its inorganic salts are concerned, is but a reproduction of the remotely ancient ocean, for the sea is the original home of all life on the globe and gave to our blood a character that long ages have not effaced and will not efface.

This imaginative comparison very cleverly suggests the germ of the old idea of œdema formation, namely, the retention by the kidney of certain inorganic constituents of the plasma, for example, salt, thereby necessitating the retention of water to maintain salt concentration at a normal level. During the course of chronic renal disease with œdema salt excretion is low, and this points to retention, but examination shows that there is no retention in the blood. On the other hand, in calculous anuria there is definite retention of salt in the blood, but no œdema. The alternative explanation, then, is that salt and water are retained in the tissues—the pre-renal deviation of Fishberg. They are not excreted in the urine because they are removed from the circulation before the kidneys can deal with them. Salt retention in the tissues occurs only as a secondary event after certain primary conditions have initiated the œdema. In other words, reduced salt excretion is the result of the œdema, not the cause of it. The tissues cannot remain œdematous unless isotonic; therefore salt is abstracted from the circulation. The more salt abstracted, the greater the amount of water the tissues can hold.

Another noteworthy point may be mentioned in passing. Ingestion of sodium chloride, sodium bromide and other sodium salts encourages œdema formation, while calcium chloride and ammonium chloride are two of our most powerful agents for promoting diuresis and disappearance of œdema. The inference is that the sodium ion and not the chlorine is the main factor in the above phenomenon, of which little is understood except the fact that it exists.

The primary factors mentioned above as being responsible for governing the interchange of fluid between the capillaries and tissue spaces are said to be: (i) the pressure within the capillaries, (ii) the permeability of the capillary wall, (iii) the colloid osmotic pressure.

Fishberg utilizes these three factors to explain the œdema occurring in Bright's disease, and in consequence postulates three distinct varieties. With slight variation of explanation, they are as follows:

1. *Cardiac Œdema.* Cardiac œdema is due to the increase fo capillary pressure from myocardial insufficiency. Further, the deficient circulation deprives the capillary wall of the required amount of oxygen, with consequent injury and increased permeability.

2. *Nephritic Œdema.* Nephritic œdema is due to increased permeability of the capillary wall, due to the action of toxins of bacterial origin.

3. *Nephrotic Œdema.* Nephrotic œdema is due to decreased colloid osmotic pressure, due to loss of albumin in urine. Variations in the albuminuria-œdema ratio are explained by the accessory factor of variation in the rate of protein regeneration. This rate, for example, is low in malnutrition, which in Bright's disease is often marked.

This hypothesis may not be generally accepted; but it is primarily attractive because it emphasizes the therapeutic possibilities, and whether it is scientifically right or wrong, the fact remains that there are varieties of œdema in Bright's disease which definitely demand different methods of treatment. Further, any conception that offers hope of affording relief acts as a foil to the fatalism that Bright's disease engenders, and as such is worthy of serious consideration before being condemned or discarded.

Glomerulonephritis.

Glomerulonephritis must be regarded primarily as an acute inflammatory lesion involving the capillaries of the glomeruli. Any conception of its beginnings, its evolution and its terminations is based on an understanding of the renal elements. The unit of structure of the kidney is the glomerulus with its capillary loops and the tubule with which it is connected and whose blood supply first passes through the glomerulus. Any toxic agent filtered out by the glomerulus reaches the tubule in a concentrated form, and therefore any glomerular damage is most likely to be followed by tubular damage of some degree. Further, any inflammatory lesion in the glomerulus secondarily causes some degree of degeneration of the tubular epithelium through interference with its normal blood supply. It is the complexity of the results possibly arising from the initial glomerular damage, that makes this part of the subject of Bright's disease so difficult to classify. For example, if the glomerular mischief is mild, it may escape notice, but it may be severe enough to cause a degenerative tubular lesion resembling a primary lipoid degeneration. And it is this possible stage in the evolution that has given rise to such nomenclature as glomerulonephritis of nephrotic type, or that has even made many observers doubt the existence of a primary lipoid nephrosis. Furthermore, if the latent stage is further prolonged and the condition does not come under observation until vascular degeneration is pronounced, it may simulate a primary arteriosclerotic condition. The difficulty in differentiation of types is therefore frequently inversely proportional to our knowledge of the early phases of the disease.

There is one important factor deserving of special emphasis and that is the number of renal units ordinarily active at any one time. It has been shown experimentally that under normal circumstances as many as 75% or more of the glomeruli may not be filtering. This is a probable explanation of why the disease is sometimes focal and sometimes diffuse. Further, when glomeruli are damaged and undergo atrophy, the tubules connected with healthy units undergo hypertrophy, even the corresponding glomeruli taking part in the process, and it is the completeness of the compensatory mechanism that possibly determines the prognosis.

Diffuse glomerulonephirtis is too familiar to require description. The acute attack is not a bacterial invasion, but a toxic spoiling of capillaries, probably generalized, but with greatest damage in glomerular capillaries. That it occurs after the primary infection has died down suggests the possibility that there is some allergic factor in it. The body is gradually immunized and when immunization is sufficiently established, the rapid lysis of the remaining organisms liberates sufficient toxin to cause the catastrophe. Chronic diffuse glomerulonephritis is really the least chronic of all forms, as 80% of patients die before the age of forty. It is a disease characterized by a gradual progressive and inevitable failure of renal function, with cardiovascular changes usually following at a discreet distance, and rarely close enough to modify to any great degree the final picture of uræmia.

The acute focal form also needs no description. It is mostly a disease of childhood which has a comparatively short course and which never produces any clinical or biochemical evidence of renal failure.

There now remains the large group of cases of glomerulonephritis in which the evolution is obscure. First, there is the type that simulates a pure nephrosis before passing on to the final stage. The early process is often latent, particularly in those cases confused with those of the nephrotic types. Not infrequently there is some history of an acute infection of the upper respiratory tract. The outstanding clinical features, when the condition usually comes under observation, are marked albuminuria and œdema. It is difficult or impossible to establish any renal inefficiency by biochemical means, and cardiovascular changes are as a rule clinically absent. Hence the confusion. Clinical observation, perhaps over years, finally settles the diagnosis. The initial lesion is a glomerulitis and is moderate, while the tubular degeneration is secondary and severe. Why such an evolution occurs is unknown. There is finally in this group the picture of renal failure; though vascular changes and cardiac failure and possibly intercurrent secondary infections frequently take their toll.

As an example of another difficulty a case history might be quoted.

A male patient, aged forty-three years, in September, 1931, complained of the usual syndrome following an influenzal attack three weeks previously. He said his urine had been very dark. Examination revealed no abnormality. The urine was clear chemically and microscopically. The systolic blood pressure was 130 and the diastolic pressure 80 millimetres of mercury.

Two months later, in November, he complained of a sore throat of four days' duration, and again passed dark urine. Examination revealed a follicular tonsillitis. The urine contained a small amount of blood. A heavy cloud of albumin was present. The specific gravity was not recorded. The blood pressure was the same as before. The fundi were normal. A suppurative *otitis media* was the immediate sequela.

A month later, in December, the urine had not completely cleared; it continued to contain blood. Albumin, pus cells and motile bacilli were present. The patient complained of weakness, shortness of breath and anorexia.

Kidney function tests were performed.

In the dilution and concentration test the amounts were three and a half, three, and three and a half ounces, and the specific gravity of all specimens was 1·010.

In the urea concentration test the second specimen had a concentration of 1·5%, the third, 1·6%.

The blood urea was 109 milligrammes *per centum*.

At the end of January, six weeks later, the urea concentration figures were 1·5%, 1·6% and 1·7%. The blood urea was 60 milligrammes *per centum*. The urea concentration factor was 18. The percentage of average normal function was 28·15. The urine contained red blood corpuscles, a few leucocytes and cocci. Cellular and granular casts were present.

In May the tonsils were removed by diathermy. The subsequent history showed that the urea concentration remained about the same. The systolic blood pressure in May was 122 millimetres of mercury and the diastolic pressure 68 millimetres, and in June the figures were 140 and 70 millimetres.

The urine at the beginning of July contained no blood, but a moderate deposit of albumin. The systolic blood pressure was 135 and the diastolic pressure 70 millimetres of mercury. The patient was back at work and feeling well.

An ordinary bacterial infection and a focal type of glomerulonephritis appeared the most likely conditions in the early stages, but the subsequent history disproved one, and the biochemical findings disposed of both. This history serves as a peg on which to hang another of our difficulties. It probably belongs to the large group which we do not see until the middle phase of the evolutionary process. There is little or no previous history to guide us. There is a long latent period with repeated attacks of glomerulitis affecting a few glomeruli at a time, never severe enough to warrant attention, but slowly and inevitably reducing the number of effective units. The gradations of glomerular destruction have been histologically proved, and the microscope has conclusively shown that macroscopical evidence may be not only inaccurate, but definitely misleading. Actually, the end picture, though perhaps long delayed, approximates to the ending of the diffuse type. The early stages are indeterminate because we are endowed with so much reserve renal tissue, and successive attacks are required to overcome, firstly, this natural reserve, and secondly, the compensatory hypertrophy of the structural units that survive after each attack.

Nephrosis.

For the remainder a few generalizations must suffice. First, the nephroses. They are characterized by primarily degenerative lesions of the specialized renal tissue, chiefly the tubules. There is little or no structural alteration in the glomeruli, and there is an absence of any histological signs of inflammation, and further, a very greatly diminished liability to fibrosis. A mild type of tubular degeneration occurs at the height of severe infections, specific fevers, or during the course of diabetes in younger persons where vascular changes have not yet occurred. The amyloid type is more the end result of some prolonged suppurative process, where the primary condition overshadows the renal condition subjectively and objectively. Quite probably, severe tubular damage frequently heals completely, because the tubular epithelium has considerable power of regeneration, whereas the glomerular structure has a very limited regenerative capacity.

The clinical condition now referred to as chronic nephrosis is essentially a disease of childhood and young adult life, and its supposed ætiology is a variegated assortment that so often goes with complete ignorance. Syphilis is credited with being a common factor, and other infections have a minor share of the blame. Clinically, chronic nephrosis presents an intense and persistent albuminuria, a marked œdema, increased blood cholesterol, lowered basal metabolism, with no signs of cardiovascular disease and no evidence of renal inefficiency. The urine is of high specific gravity and contains abundant casts and doubly refractive lipoid bodies. It is one of the rarest diseases and cannot be diagnosed on its main symptoms without months, perhaps years, of observation. Glomerulonephritis in one of its perplexing forms may simulate it accurately, and this fact has caused endless confusion and controversy. The key fact appears to be albuminuria, possibly the result of a general disorder of metabolism; the other symptoms may all be secondary to this loss. So far, no one knows. Patients usually recover, it is said, after varying periods, perhaps of years, though secondary infections, particularly pneumococcal, may terminate the history prematurely.

Arteriosclerosis.

Regarding the arteriosclerotic group, it must be remembered that arteriosclerosis (an omnibus name is Clifford Allbutt's description of it) includes such varied pathological states as atheroma of the aorta, pipe stem radial arteries and the arteries of essential hypertension. With the latter alone is Bright's disease connected. The primary change in essential hypertension is an elevation of blood pressure from an unknown cause. "The very term essential hypertension", says Fishberg, "is a confession of ignorance—and this is its chief virtue." A compensatory hypertrophy of the left side of the heart and of the muscular elements of the whole arterial system follows. In the kidney the small arteries and arterioles so affected have a special disposition to show degenerative changes, with a narrowing and obliteration of their lumina.

The secondary result is atrophy of renal elements from deprivation of blood. Actually, the arteriosclerotic patient rarely dies of renal failure, and

though many die in coma with convulsive seizures, failure of their renal function is not the cause, and their deaths should not be called uræmic. Cardiovascular degeneration in over 90% of cases outdistances degeneration of renal tissues, and the vanishing point of renal reserve is not reached. Death occurs from failure of cardiac reserve or from cerebral vascular accidents.

The whole problem of the cardiovascular renal triad is still a trinity of mystery to physiologists. But as knowledge slowly accumulates, there is a realization that it is futile, from the point of view of progress, to classify Bright's disease according to the final macroscopical pictures which the passage of years has painted, when there is complete ignorance of the tortuous processes by which it reached its final stage. It is just as sensible as attempting to compute the individual values of three unknowns, when only their sum total is given.

A final commentary may be given in the words of Clifford Allbutt, distinguished alike for the wealth of his knowledge and the graceful and elegant manner in which he expressed it.

> The problem is one of the most alluring and most baffling in pathology, alluring perhaps by its very subtlety. The ways of approach are strewed with guesses, presumptions and conjectures, the untimely and sterile fruitage of minds which cannot bear to wait for the facts, and are ready to forget that the use of hypothesis lies not in the display of ingenuity, but in the labor of verification.

THE FUNDUS IN NEPHRITIS.[1]

By J. C. DOUGLAS, L.R.C.P. and S. (Edinburgh),
Honorary Oculist, Ballarat Hospital, Ballarat.

THE fundus frequently presents a very characteristic group of changes in kidney disease. The fundus changes, when they do occur, are called albuminuric retinitis or renal retinitis.

Albuminuric retinitis does not occur in the ordinary type of acute nephritis. It occurs frequently in those primary acute forms known as the nephritis of pregnancy, scarlatinal nephritis, and the nephritis of trench warfare. It is not common in chronic parenchymatous nephritis, occurring in 15% to 30% of cases. It occurs in 40% to 50% of cases of chronic interstitial nephritis.

Renal retinitis is twice as common in men as in women, because chronic nephritis is twice as frequent in men. The presence of renal retinitis may be taken as evidence that the kidney is passing beyond power of recovery. It is a guide, therefore, to a bad prognosis. Patients with chronic nephritis and albuminuric retinitis generally die within two years. Indeed, 65% will probably die within one year. However, patients whose retinitis is associated with arteriosclerosis, pregnancy, and scarlet fever must be excluded from this statement of prognosis.

[1] Read at a meeting of the Victorian Branch of the British Medical Association, July 23, 1932.

Renal retinitis shows itself subjectively by dimness of vision; it rarely causes complete blindness, as in uræmic amaurosis. It is nearly always associated with a much increased systolic blood pressure — 200 millimetres of mercury or more. Severe headache and albuminuria are the other principal symptoms. If the patient recovers from the nephritis, then the retinitis does not tend to return in subsequent attacks.

Just as there are two types of nephritis, acute and chronic, so there are two types of fundus picture, the toxic and the vascular.

The more acute or toxic form of nephritis produces three characteristic changes in the eye grounds. These are large white exudates called cotton wool patches, hæmorrhages of flame shape, small in size and not numerous, and a moderate degree of optic neuritis or papillitis. The white exudates are fibrinous, the hæmorrhages are due to diapedesis through the capillary walls and not to rupture of vessels, the optic neuritis varies from an angry, blood-streaked, toxic disk to a diffuse, cloudy swelling of greyish colour. In chronic kidney disease the white exudates are smaller, brighter, solid-looking, sharp-edged, and may form a star figure at the macula. This consists of discrete white dots and rods arranged radially or fan-like, and is characteristic but not pathognomonic. The hæmorrhages are more numerous and flame-shaped or round. Vascular changes occur, such as engorgement of the veins, with kinking, alteration in the reflex of the arterial walls, and other signs of high tension. Optic neuritis is less frequent.

With the subsidence of the nephritis the fundus changes disappear, but defective vision is often left as an unfortunate sequel. Particularly does this occur after the albuminuric retinitis of pregnancy.

Of all patients with kidney disease only some 20% to 30% will have retinitis. The majority of patients with renal disease, therefore, have no retinitis. Most of the patients who do have retinitis also have vascular disease in the retina. There is no constant relationship between the retinitis and the vascular disease, however, and the vessels may be quite normal.

While the retinitis is probably due to a toxin circulating in the blood stream, yet the degree of retinitis bears no fixed relationship to the nature or severity of the renal mischief. Retinitis is nearly always bilateral in nephritis, while in arteriosclerosis it is unilateral in 50% of the cases.

If I were to contrast chronic parenchymatous with chronic interstitial nephritis, I should say that in the chronic parenchymatous nephritis you see in the fundus cotton wool patches, retinal œdema, retinal detachment, sometimes few hæmorrhages, and no star figure. In chronic interstitial nephritis, on the other hand, you find the retinal vessels sclerosed, many hæmorrhages, and a star figure at the macula.

Lastly, the essential relationship in the ætiology of the retinitis of chronic nephritis, arteriosclerosis

and diabetes is closer than was formerly supposed. The basis of the pathology of all three is probably the same, and high blood pressure and albuminuria are common to all.

SOME ASPECTS OF THE RENAL COMPLICATIONS OF PREGNANCY.[1]

By RAYMOND GRIFFITHS, M.B., B.S. (Melbourne), F.R.C.S. (Edinburgh), F.R.A.C.S.,
Honorary Obstetrician, Ballarat Base Hospital, Ballarat.

BEING much interested by James Young's[1] views on the nature and sequelæ of the renal toxæmias of pregnancy, I have attempted to see if the record of cases in the Ballarat Hospital would lend them any support.

Unfortunately, sometimes the records were far from complete, and my own follow-up of the patients still more incomplete, as I have not yet written to any of them to find out their present condition. I have relied on their past histories, and the records of any readmissions to the hospital.

Eliminating a large number of patients whose urine was charted as having contained albumin, but who presented no toxæmic symptoms, and for whom no special treatment was instituted, there were, from September, 1926, till June, 1932, twelve patients with eclampsia, and forty-eight other toxæmic pregnancies occurring in forty-five patients. There were also three patients with accidental hæmorrhage who exhibited albuminuria; these are not included in this series.

Eclampsia.

Of the twelve patients with eclampsia, one, the twelfth, died; of the thirteen babies, five died—a maternal mortality of 8·3% and a fœtal mortality rate of 38·8%.

Following Eden, cases of eclampsia may be divided into mild cases or severe. Eclampsia is said to be severe if the patient exhibits any two severe signs or symptoms, for example, deep coma, many fits, temperature over 39·4° C. (103° F.), pulse rate over 120 per minute, "solid" albuminuria, and a systolic blood pressure of over 200 millimetres of mercury.

On this basis four cases could be classified as severe, and in this group there was one maternal death (a mortality rate of 25%) and there were three fœtal deaths among five babies (a mortality of 60%).

In the eight cases classified as mild no mothers died, and but two babies (a fœtal mortality of 25%).

There were two patients with *post partum* eclampsia, neither of whom died. One case gave a history of previous toxæmic pregnancies, the first and fourth pregnancies resulting in still-born children, and the fifth as well as the present one (the sixth) being eclamptic.

One patient whose eclampsia was classified as mild, was admitted nearly three years later with severe pre-eclamptic symptoms, and a subsequent pregnancy five months later was terminated by therapeutic induction at three months.

Two patients have subsequently had normal pregnancies. The remainder of the patients have not been readmitted to the hospital.

Of the twelve patients, eight were *primiparæ*. The treatment adopted, with a few minor modifications, was that used at the Rotunda Hospital, Dublin. Morphine 0·015 to 0·03 gramme (one-quarter to one-half a grain) was given on admission to hospital, and was repeated in doses of 0·015 gramme (a quarter of a grain) with each fit until the fits had been controlled or until 0·12 gramme (two grains) had been given. Colonic lavage was instituted and repeated in six hours if the patient's condition was not satisfactory. *Mistura sennæ composita* 60 cubic centimetres (two fluid ounces) was left in the rectum with 600 cubic centimetres (a pint) of bicarbonate of soda solution. If the patient was conscious and not vomiting, an aperient such as *pulvis jalapæ compositus* was administered. If the patient was vomiting, the stomach was washed out and 60 cubic centimetres (two fluid ounces) of *mistura sennæ composita* were left in the stomach.

When the condition was apparently controlled chloral hydrate 1·2 grammes (twenty grains) and potassium bromide 1·2 grammes were administered orally, at first every four hours and later two or three times a day. The usual general measures, such as placing the patient in a quiet darkened room, avoiding unnecessary manipulation, turning her on her side to allow of the escape of mucus and saliva from the mouth, and the provision of a gag to prevent biting of the tongue during seizures, were adopted as a routine. Hot linseed poultices were applied to the loins. Veratrone was not used; venesection was performed in two cases, once only as a last resort. Labour was induced in one case by Watson's method. In the other *ante partum* cases labour commenced spontaneously within three or four days of admission. If labour had not commenced within a week following the control of the convulsions, I would consider induction advisable, by Watson's method, or by the introduction of a stomach tube into the uterus. I would consider Cæsarean section only in certain cases of a fulminating type in a *primipara* who was not in labour.

The history of the fatal case deserves to be placed on record.

Mrs. G., thirty-four years of age, who had had one normal pregnancy previously, was admitted on May 14, 1932, with a history of being unconscious since early morning; she was eight months pregnant, and there had been no fits. She had fallen on the previous day, but had not sustained a head injury. On admission she was unconscious, the systolic blood pressure was 134 millimetres of mercury, and the urine became solid on boiling. That evening the patient had three fits in quick succession and shortly afterwards was delivered of still-born twins.

[1] Read at a meeting of the Victorian Branch of the British Medical Association, July 23, 1932.

The usual treatment was instituted. The next day the patient displayed some irritability on being touched, but could not be roused. As she did not respond to treatment, lumbar puncture was performed and clear fluid under increased pressure was withdrawn. This produced no improvement, but showed that the presence of a head injury was unlikely. Venesection was also performed without apparent benefit. The condition remained the same on the next day. On the fourth evening after admission there were three more fits in quick succession, and on the following morning the patient died. Dr. Douglas reported on the first day that each eye showed a retinal detachment.

A limited *post mortem* examination showed that the liver and kidneys were macroscopically normal. The cortical vessels of the brain were engorged, but no other abnormality was found.

Preeclampsia.

Forty-eight cases of toxæmia occurred in forty-five patients who required treatment for albuminuria with symptoms of renal toxæmia without convulsions. Of these forty-five, twenty-five were *primiparæ*. No maternal deaths occurred in this series, but there were nine fœtal deaths—a fœtal death rate of 18·7%.

Including the twelve cases of eclampsia, there were sixty cases of renal toxæmia in a total of 867 admissions to the maternity ward—a percentage of 6·92%. Cases of eclampsia formed 1·38% of the total. Of the patients who had no convulsions, eight gave an abnormal previous or subsequent history.

1. B.A., aged nineteen, in her first pregnancy, had severe albuminuria. Her second pregnancy five years later resulted in an incomplete abortion.

2. V.S., aged thirty-six, had albuminuria in her third pregnancy. She had had two miscarriages previously, at three and four months respectively.

3. H.W., aged thirty-one, had an induction of labour in her first pregnancy; the reason is unknown. Labour was induced in her second, third and fourth pregnancies because of albuminuria. In her fifth pregnancy albuminuria occurred. The sixth pregnancy resulted in miscarriage.

4. R.M., aged forty-one, had albuminuria in her sixth, seventh, eighth and ninth pregnancies (one *ante partum* hæmorrhage). The tenth child was still-born at full time. In the eleventh pregnancy albuminuria occurred.

5. J.O'L., aged twenty-nine, had albuminuria in her third pregnancy. Her fourth pregnancy resulted in an incomplete abortion.

6. H.W., aged twenty-five, had a miscarriage and albuminuria in her first pregnancy at six months. In her second pregnancy preeclampsia was present.

7. M.F., aged twenty-four, had eclampsia at eight and a half months in her first pregnancy. In her second and third pregnancies preeclampsia occurred, the last at six and a half months.

8. F.M., aged twenty-three, had mild albuminuria at full time in her first pregnancy. In her second pregnancy albuminuria occurred.

Two patients were readmitted with normal pregnancies.

From the past and present histories and physical signs eight patients were thought to be suffering from chronic nephritis complicated by pregnancy. Nine pregnancies in these eight cases showed a fœtal mortality of 33%. In thirty-nine pregnancies occurring in thirty-seven patients, judged to be examples of pregnancy toxæmia, there were six fœtal deaths—a fœtal mortality rate of 14·9%.

Prognosis.

Although this series gives my opinion little support, I take the view that a toxæmic pregnancy renders the patient more likely to have in any future pregnancy abnormalities such as albuminuria, accidental hæmorrhages, miscarriages *et cetera*. In many cases without doubt there is a progressive renal deterioration with each pregnancy.

If, following delivery, the albumin disappears within ten to fourteen days, if the blood pressure falls to normal, and if the response to renal function tests is good, I advise against a future pregnancy for two years, and insist that in a future pregnancy the patient should place herself under observation as early as possible.

Where there have been two or more toxæmic pregnancies, I advise the patient not to become pregnant again.

In such a case, should the child have been lost in every pregnancy, and if the patient is desperately anxious to have a child, I would allow of a pregnancy after a three-year interval, provided that the response to renal function tests was good. In such cases I endeavour to remove any sources of focal sepsis and subject the patients to a very close antenatal supervision.

Prevention and Treatment.

The prevention of eclampsia is in the great majority of cases purely a matter of efficient antenatal supervision. We examine the urine in every case once every four weeks during the first six months and thereafter once every two weeks. Since Professor Browne, of University College Hospital, has drawn attention to the fact that in many cases there is a progressive rise in the systolic blood pressure some time before the appearance of albumin in the urine in toxæmic cases, we have made a practice of estimating the blood pressure at each visit. From personal experience I cannot give an opinion of its value. In several cases the systolic blood pressure rose steadily during the last month of the pregnancy and fell rapidly to normal after the confinement, without albuminuria or toxæmic symptoms developing. We have dieted such patients and ordered mild eliminative treatment; whether the treatment prevented the onset of toxæmia or was entirely unnecessary must remain an open question.

Any patient developing a trace of albumin in the urine or a raised blood pressure is asked to report every week for further examinations; dietary restrictions are imposed and mild eliminative treatment is ordered.

Each patient, on her first visit to the hospital, is warned of the symptoms of impending toxæmia—headaches, epigastric pain, vomiting, swelling of the hands or face, dimness of vision and oliguria—and is given a printed sheet of instructions and told to report immediately if any untoward symptoms appear.

Treatment of Preeclampsia.

If symptoms are present or the amount of albumin is more than a trace, the patient is ordered into

hospital. In the milder cases a carbohydrate diet is given, with free fluids; the bowels are kept open freely with *pulvis jalapæ compositum* four grammes (one drachm) once or twice a day.

In the severer cases nothing but water is allowed for twenty-four hours; after that, if response to treatment is satisfactory, a carbohydrate diet is given.

Pulvis jalapæ compositum four to eight grammes (one to two drachms) is given by mouth, and a colonic lavage is ordered. This is repeated in eight, twelve or twenty-four hours, varying with the severity of the condition and the response to treatment. A mild diuretic and diaphoretic mixture is ordered. Except in the milder cases, I think that pregnancy should be terminated when the toxæmia has been controlled. If the child has at that stage a poor chance of survival, it seems to be permissible to continue treatment for ten days or a fortnight, when treatment is effective in controlling the disease.

Indications for Induction of Labour.

Rising blood pressure, severe headache, epigastric pain, and diminution in the amount of urine while the patient is under treatment are, I think, absolute indications for terminating the pregnancy. The presence of albuminuric retinitis is also regarded as an indication for induction. The longer conservative treatment is persevered with in the face of a poor response to treatment, the greater seems to be the risk of permanent damage to the kidneys.

Induction of Labour.

In inducing labour we try first Watson's method, if there is no need for haste. If this fails or if a more rapid method is needed, a stomach tube is inserted into the uterus. It is important not to relax treatment once labour is in progress.

Reference.

(1) James Young: "The Prognosis and Treatment of Eclampsia and Albuminuria", *The British Medical Journal*, January 19, 1929, page 91.

RENAL FUNCTION TESTS AND THEIR APPLICATION TO SURGERY.[1]

By G. R. DAVIDSON, M.B., F.R.C.S. (Edinburgh), F.R.A.C.S., *Ballarat.*

THE essential function of the kidney is the formation of urine from the blood plasma by the concentration of certain substances already present in the blood, some only when in excess of a certain value, others always in proportion to their amounts. Probably the earliest attempts at any estimation of limited kidney function were those of Rayer. He noted the absence in nephritis of that familiar odour which is normally present in the urine after eating asparagus, and regarded this absence as an indication of renal dysfunction. However, renal function tests as we know them now are a comparatively recent development, and for all practical purposes have been evolved during the last twenty or thirty years.

The ideal functional test should, I think, fulfil the following conditions:

1. It should be simple and the results should be simple to interpret.

2. It should indicate within narrow limits the constant amount of work performed by all normal kidneys under normal conditions.

3. It should indicate constant variations in function when constant abnormal conditions are present.

4. It should afford a means of estimating the latent or reserve power which can be utilized by the kidney under strain.

5. It should be applicable without injury to the patient or without exerting any call or strain on the kidney itself.

A great number of tests have been elaborated during the last fifteen years or so. They are all based on a more or less imperfect knowledge of renal physiology, and the information they give is all nearly parallel.

Factors over which we have no control often render the interpretations of the various findings difficult. The chief of these factors is renal inhibition, and the commonest type of this is a reflex inhibition. Failure of renal activity after urethral catheterization is a common example.

At the same time, a pronounced polyuria associated with deficient concentrating power may occur after ureteral catheterization. Other factors, such as mental states, toxins, chemicals, pressure and obstruction, may play their part in producing renal inhibition. Varying periods of activity and inactivity have been demonstrated for individual units as well as for whole portions of the kidney. Richards[1] has shown this in his work on the glomeruli of the frog's kidney. Apparently the glomeruli do not always work to their full capacity, and these units themselves can contribute towards an increased total function by an increase in their activity. These possibilities of reflex inhibition or stimulation of function constitute a definite limitation to the value of studies of renal function, rendering necessary at all times a proper appreciation of each individual patient's clinical condition. Functional tests serve two purposes, the determination of total function and of relative function, and there are three groups of renal conditions for consideration—medical diseases, surgical diseases, and renal dysfunction due to lower urinary tract obstruction. The so-called medical lesions are usually bilateral, the surgical conditions are frequently unilateral, and even when they are not, an estimation of the relative efficiency is very valuable. Renal dysfunction due to lower urinary tract obstruction is characterized frequently by a remarkable improvement after the obstruction is relieved. It is well to remember that all renal function tests must be

[1] Read at a meeting of the Victorian Branch of the British Medical Association, July 23, 1932.

regarded as purely empirical, and that they indicate renal work for the period of the test and that period only. They give no indication of what the kidney has done nor of what it will do. They fail to indicate the degree of, or absence of, renal reserve.

Tests commonly used fall into two big classes, tests of excretion or concentration and tests of retention.

In the majority of cases combinations of these tests are used, depending often on individual preference. It is never suggested that one test is all sufficient or that it renders unnecessary the use of other methods.

In the time at my disposal it is impossible to do more than make a few remarks about some tests in common use and then to discuss briefly their clinical application.

Concentration and Dilution Test.

I shall first deal with the concentration and dilution test, or water test, which is simple and which is probably not used as frequently as it should be. It is actually a measure of the power of the kidneys to concentrate urine and also, I think, one that gives some slight indication of the reserve power of the kidneys.

It may be carried out as follows:

The night urine is collected from the patient at 10 p.m. and 5 a.m. At 6 a.m. one and a half pints of water or weak tea are given, and hourly collections of urine are made until 11 a.m. The volume of each specimen is measured, the specific gravity is estimated. The normal figures are fairly constant. The night specimen should possess a high specific gravity, 1·025 or higher, and should be small in volume. Of the pint and a half of water given, between twenty-five and thirty-five ounces should be excreted within four hours, and the maximum quantity should be passed in the second hour specimen, which should also possess the lowest specific gravity, 1·003 or lower. Thus a range of specific gravity of about 17 to 22 is considered within normal limits.

Slight and moderate impairment of function is shown by an increase in the volume of the night urine and a diminution of its specific gravity, and also an output in the four hours after the fluid, greater than thirty ounces.

According to Hunt,[2] this excessive water secretion is not due to any existing polyuria, but appears to be in the nature of an irritability of a kidney which is pathologically sensitive to the stimulus of a sudden excess of fluid. With kidneys that are more damaged a diminished secretion occurs, and if the volume excreted in the four hours is less than twenty-five ounces, it is an indication of renal impairment. The less the total four-hourly volume, the more do the specific gravities of each hourly specimen approach each other. Equality of volume of the specimen with a fixation of the specific gravities at about 1·010 is indicative of the gravest renal impairment and is frequently the state of affairs met with before an attack of uræmia.

It is well to remember that the results of this test are of little value when œdema is present, whether cardiac or renal. The patient should be in bed, to obviate, as far as possible, fluid loss from perspiration. The question of nervous inhibition should never be forgotten, and it is sometimes necessary to repeat the test if this is suspected.

Urea Concentration Test and Blood Urea Estimation.

The urea concentration test and the blood urea test are in very common use, and I do not propose to do more than mention a few points in connexion with their use.

One of the chief disadvantages of urea test is that urea is a diuretic, and a polyuria naturally produces figures that are too low. If the volume of the second specimen is over 180 cubic centimetres it is wiser to repeat the test. The presence of much blood in the urine tends to upset the results.

Frequently the estimation of the blood urea is carried out in conjunction with this test. It is well to remember that the blood urea estimation should be carried out before the ingestion of the urea.

Complicated formulæ overburdened with corrections, such as those of Koranyi or Ambard, have little if any real clinical value.

With regard to the blood urea test, it is worth remembering that three-quarters of the total renal tissue must be extirpated in animals before the blood urea rises.

A high blood urea is not specific evidence of renal failure, as it may be found when there is excessive loss of fluid from such conditions as sweating, diarrhœa, or high intestinal obstruction. Apart from circumstances such as these, one can generally assume that there is gross renal damage associated with a definitely raised blood urea.

Dye Tests.

Dye tests are used most frequently in comparing the efficiency of one kidney against that of its fellow. There are two substances in common use today, namely, indigo-carmine and phenolsulphonephthalein. The indigo-carmine test is not in any way quantitative. There are two points to be noted: first, the time of appearance of the dye, and secondly, the depth of the coloration during active excretion. The average time for the appearance of the dye after intravenous injection is four minutes. A late appearance, say, up to six minutes, should be atoned for by a deep coloration at the peak period of elimination. An appearance after seven minutes should cause one to regard that kidney with grave suspicion.

The chief advantages of this test are quickness, simplicity, and the fact that ureteric catheterization is unnecessary.

The phenolsulphonephthalein test is used extensively in America. This drug, whether given by mouth, subcutaneously, intramuscularly or intravenously, is practically all excreted by the kidneys.

Geragthy, who first introduced this test, stated that the capacity of picking out the molecules of phenolsulphonephthalein from infinitely dilute solution (one in 10,000,000) and passing them on to the secretion in comparatively concentrated solutions is a function specific to the kidney.

The test may be used both qualitatively and quantitatively to test not only the total renal function, but also to compare the function of one kidney with that of its fellow. After intramuscular injection, the dye takes about ten minutes to appear, and after intravenous injection about two to four minutes. Of the dye 40% to 60% is excreted in the first hour and 20% to 25% in the second. In practice the specimens taken at the end of the first and second hours, after suitable dilution, are compared colorimetrically with a standard solution of phthalein in a Dubosq colorimeter and the percentage of dye excreted is calculated.

The disadvantages of this test are, first, the difficulty of obtaining a true reading on the colorimeter, and secondly, in the case of the estimation of separate renal function to get over the difficulties of renal inhibition, catheter blockage, and leakage alongside the catheter. The last mentioned can be obviated to a certain extent by using flute-ended catheters.

Another disadvantage is the long period for which ureteric catheters have to be left *in situ*. I have not had a great deal of experience of this test, but regard it as valuable when the indigo-carmine test gives a doubtful or inconclusive result. It is undoubtedly a more sensitive test than the latter.

Excretion Urography.

Lately investigations have been made into the use of substances used in intravenous or excretion urography as a measure of renal function.

Three methods suggested themselves: (i) a study of the density of the shadow thrown by the contrast substance; (ii) the estimation of the eliminated substance itself, or combined iodine in the substance; (iii) a study of the specific gravity of the urine after the injection of the "Uroselectan" or corresponding substance.

With regard to the first, we know that visualization is "negative" in pronounced kidney insufficiency, but we also know that in the presence of peripheral obstruction one may obtain a visual result to a functional test even in the presence of pronounced kidney insufficiency. It is necessary, then, if any information is to be obtained from the study of pyelograms after injection of "Uroselectan" or similar substances, that there should be a proper appreciation of any factors causing obstruction.

With regard to the second and third, I think it is sufficient to say that recent experimental work tends to show that in both normal and impaired renal function the excretions of "Uroselectan" as estimated by the iodine content of the urine runs more or less parallel with the urinary volume output.

In normal persons there is a diuresis following its administration which is at a maximum in the first hour specimens. By the third hour this diuresis has passed off and by the third to the fifth hour the greatest quantity of iodine has also been eliminated. The maximum concentration of iodine corresponds generally in time with the maximum specific gravity observed.

To summarize:[3] This experimental work raises the question as to the value of the measurement of volume output and specific gravity following the injection of "Uroselectan", as a measure of kidney function without resorting to the difficult and lengthy estimation of iodine. It is possible that the method will be developed in the future.

Von Lichtenberg states that the "Uroselectan" group is eliminated principally through the glomerulus, and he claims that the method is of great value in detecting glomerular damage.

Clinical Considerations.

From the surgical point of view it is necessary to evaluate both unilateral and bilateral renal function. When the question of nephrectomy is under consideration, it is obviously of vital importance that the patient should be known to possess two kidneys, one of which is capable of "carrying on" after the removal of its fellow. In bilateral renal disease it is again of paramount importance to appreciate as fully as possible the relative efficiency of each kidney. In these bilateral cases the factor of renal counterbalance is not taken into consideration as often as it should be. By counterbalance is meant an attempt on the part of the less injured or uninjured portion to take over the work of the more injured portion. Consideration of this factor of renal counterbalance is very necessary in any conservative renal surgery. A brilliantly executed plastic operation on a unilateral hydronephrosis is often doomed to failure because stimulation to repair, even after the most highly successful treatment of the cause of the obstruction, is not so active or so great as in the case of an exactly similar bilateral condition. Surgery properly applied to cases of bilateral hydronephrosis is more frequently crowned with success, but it is obvious that the intervals between the repair operations should not be any longer than necessary. In the majority of cases the dye tests give the information that is needed.

In my experience a positive result to an indigo-carmine test has always indicated impaired function. On the other hand, very occasional cases have been reported in which a badly damaged kidney shows a good excretion of the dye. If any doubt exists, the phenolsulphonephthalein test, controlled from factors such as inhibition, leakage *et cetera*, will undoubtedly give more accurate information.

Considering total renal function, one must always remember that here especially clinical symptoms are invaluable in the diagnosis of renal efficiency, and should always be considered in the

interpretation of any tests. The main use of these tests is in prostatic conditions. The tests that we chiefly use here are the water test, the blood urea test, and the urea concentration test and factor.

Before doing a one-stage prostatectomy, I prefer that the specific gravity range after the dilution and concentration test should be little below 15, the percentage of urea in the second specimen should be 1·5% or over, and the blood urea should be below 50 milligrammes per 100 cubic centimetres of blood.

In a series of cases, which incidentally is a first series, I have performed the operation of suprapubic prostatectomy for simple prostatic hypertrophy nineteen times without mortality. I have lost one patient with prostatic malignant disease.

In a small series of cases such as this, one is hardly able to develop either a satisfactory standard technique or a high degree of surgical skill, and I think one is justified in assuming that it is only by a fairly rigid adherence to the principles that I have already enumerated, that these results have been at all satisfactory. I am quite aware that more experienced surgeons do not always demand these standards. Thompson Walker,[4] in a paper entitled "Views on Prostatectomy", written in 1924, wrote as follows:

> Some time ago I found in one of my hospitals that an able house surgeon, keeping himself abreast of current opinion, was sending up a series of cases for bladder drainage by cystotomy as a preliminary to prostatectomy. This was on account of impaired renal function, the urea concentration being 1·4% to 1·6%. Thus a very simple rule of thumb was developing, namely, urea below 2%, drain the bladder and wait, above 2%, single stage prostatectomy. This arbitrary rule did not appear to me to be justified, and I decided that unless some other factor contraindicating operation was present, I would operate in all cases when the urea concentration test was above 1%. The results have justified this procedure.

He goes on to mention that he has frequently operated successfully on patients with blood urea from 60 to 80 milligrammes per 100 cubic centimetres. He concludes by stating that laboratory figures of the renal function tests form only one factor in estimating the prognosis for operation among many, which include the general build and constitution of the patient, the weakness and soundness of the organs, the experience of the operator, and, above all, the experience of those who have the immediate care of the patient after operation.

In conclusion I wish to state that I am quite aware that I have said nothing that is new and nothing that has not been said before. My only apology is that the subject is one of vast importance, a proper appreciation of it having been the means of saving thousands of lives.

References.

(1) A. N. Richards: "Kidney Function", *The American Journal of the Medical Sciences*, Volume CLXIII, 1922, page 1.
(2) T. C. Hunt: "Water Test of Kidney Function", *The British Journal of Urology*, March, 1931, page 26.
(3) D. P. Cuthbertson and A. Jacobs: "Intravenous Urography: Preliminary Observations on the Recovery of Iodine as a Test of Renal Function Following the Injection of Uroselectan", *The British Journal of Urology*, March, 1932, page 36.
(4) Thompson Walker: "Views on Prostatectomy", 1924.

Reports of Cases.

AN UNUSUAL CASE OF ACTINOMYCOSIS.

By the late A. V. M. ANDERSON, M.D., B.S.,
Sometime Honorary Consultant Physician, The Alfred Hospital, Melbourne,

AND

ALFRED J. TRINCA, M.D., F.R.C.S. (England),
Honorary Surgeon to Out-Patients, Alfred Hospital; Consultant Pathologist to the Baker Institute of Medical Research, Melbourne.

THE following fatal case of actinomycosis is of interest on account of its long duration and the apparent period of quiescence or arrest during a period of eleven years.

V.F., female, single, was first seen by one of us (A.V.M.A) on January 15, 1915. She was a delicate looking girl of seventeen years and had been employed in an ammunition factory. Five months previously she had developed a cough and had been seen by her usual medical attendant, who diagnosed right-sided pleurisy and pericarditis and ordered her to bed. After a period of two to three months she went to the country for a holiday, but had to seek medical advice there, and was again ordered to bed. There had been some loss of weight (she weighed 35·1 kilograms or five stone eight pounds when first seen), with pain in the lower part of the right side of the chest, and she had also not menstruated since the beginning of her illness. The family history was very good, seven brothers and sisters being alive and well.

When examined, the patient's temperature was 38·7° C. (101·8° F.). There was definite bulging over the right lower ribs, just below the breast, with dulness on percussion over the right base and diminution of breath sounds; there was also some left-sided pleural friction. The heart's action was rapid, the position of the apex normal, and no murmur nor pericardial rub was audible. The liver seemed somewhat displaced downwards. An examination of the blood showed the number of leucocytes to be 15,360 per cubic millimetre. There was no evidence of any increase of eosinophile cells in a stained film.

The swelling of the chest appeared to involve the ribs, and the diagnosis made was either sarcoma or actinomycosis. Puncture of the swelling was advised.

In March, 1915, she became an in-patient at the Alfred Hospital. An abscess had formed at the site of the swelling in the chest. This was opened and a bacteriological examination of the pus revealed the presence of streptothrix actinomycosis. At this time an abscess had developed above the left elbow, and there was also a swelling over the trachea. Dulness, with diminished breath sounds, persisted at the base of the right lung.

She was given potassium iodide in doses increasing up to 4·0 grammes (one drachm) three times a day. A culture was made from the pus and a vaccine was prepared, which was administered for some months. She was in hospital for about a year, gradually improving all the time. Pus formed in the swelling of the arm and neck and was evacuated, the sinuses closing and then again opening.

On August 8, 1916, her weight had increased to 48·6 kilograms (seven stone ten pounds), the menstrual flow had occurred regularly, and she became a pupil at a business college. The physical signs at the right base were still present, and occasionally crepitations could be heard at the left base.

On October 2, 1917, there was some diminution in weight (46·3 kilograms or seven stone five pounds) and the physical signs in the chest had not altered much, but she felt in good health and was continuing with her work.

She was not seen again until June, 1927, when she was contemplating marriage. As the condition appeared quiescent, there appeared to be no reason why marriage should not take place. Except for a period of nine months, which had been spent in a visit to Western Australia, she had been occupied in office work and had

enjoyed good health, being able to play tennis without discomfort. She had had a little dry cough and her weight had fallen to 41·8 kilograms (six stone nine pounds). The dulness at the base of the right lung still persisted, with diminution of breath sounds, and there were some crepitations at the upper margin of the dull area. The cardiac apex beat was slightly displaced to the right, but moved with change of position.

The marriage was postponed and the patient was again seen on April 4, 1929, four days before her contemplated marriage. On account of painful menstruation, she had consulted a herbalist and had been taking medicine. The chest condition had not appreciably altered. There was a small thickening over the biceps tendon of the left arm, another lump in the right thigh above the knee, and another in the lumbar region. These suggested a papular iodide rash.

She was again seen on May 15, 1929, on her return from her honeymoon. The swelling in the left arm had increased considerably in size, while that in the right thigh had become less. Other lumps had appeared in the left leg and thigh, and an abscess had developed in the lumbar region. She was then transferred, on May 18, 1929, to the Alfred Hospital, to the care of A. J. Trinca.

On examination, she looked wasted and ill, the pulse was irregular and she had the following swellings: One in the mid-line of the back, at the level of the fifth to tenth dorsal spines; one over the left antecubital fossa; two in the left calf; and one in the front of the right thigh. The lungs showed good expansion and breath sounds were moderately good, except at the base of the right lung. On the same day the abscesses in the back and calf were opened. Characteristic actinomycotic pus escaped, from which a culture was made and a vaccine prepared. Lugol's solution, 0·6 mil (ten minims), increasing by 0·06 mil a dose daily, was given three times a day until 4·0 mils (one fluid drachm) were taken three times a day.

On May 24, 1929, her chest was examined by X rays. The right diaphragm was immobile and definitely raised, suggesting the presence of a collection of fluid under the diaphragm, while a slight elevation of the central portion of the left diaphragm suggested the possibility of a second collection. The lungs were clear.

On May 26, 1929, the wounds in the calf and back were almost healed.

On June 3, 1929, an abscess was discovered in the right foot and opened.

On June 11, 1929, vaccine treatment was commenced, a vaccine with an opacity equal to 100,000,000 bacilli to one cubic centimetre being used. This was given subcutaneously in 0·5 cubic centimetre doses every fifth day, increasing by 10,000,000 per dose, and continued for some weeks.

A sinus between the second and third toes of the left foot was syringed with ticture of iodine.

On July 23, 1929, all the old incisions were reopened and pus containing granules was found in all of them. The swelling of the left arm was incised and a large quantity of pus was evacuated. The swelling in the neck was also incised and an abscess was found tracking under the trapezius. A counter opening was made at the back. The collection of pus over the left ischial tuberosity was opened. All the wounds were packed with iodine gauze and the patient was given Lugol's solution up to 8·0 mils (two fluid drachms) three times a day.

On July 24, 1929, the gauze packs were removed under a general anæsthesia.

On August 2, 1929, another abscess was discovered at the back of the arm, just above the elbow and pointing to the lateral side of the biceps tendon. This was opened on August 5, 1929.

On October 4, 1929, the patient was given potassium iodide 1·2 grammes (20 grains) three times a day, in addition to tincture of iodine 4·0 mils (one fluid drachm) three times a day.

On October 14, 1929, the abscesses at the left axilla and the left elbow were aspirated and on October 17 the abscess in the right elbow region was opened.

On October 24, 1929, the axillary abscess was again aspirated and the patient was transferred to the Austin Hospital, where she died on January 9, 1930. Autopsy was performed on January 10, 1930. We are indebted to Dr. R. A. Willis for the following *post mortem* notes.

Post Mortem Findings.

V.K., aged thirty-one years, died at 10.30 a.m. on January 9, 1930. *Post mortem* examination was performed at 10 a.m. on January 10, 1930.

The body was that of a young, very emaciated woman. There were scars down the inner surface of the humerus of the right arm and around the elbow joint. The elbows were fixed and the hands œdematous. Similar scarring was present on the inner surface of the right leg.

The thoracic viscera in the anterior mediastinum were adherent to the sternum. The pericardium was adherent to the heart and lungs, but there was no obvious actinomycotic infiltration.

The right lung weighed 240·5 grammes. Numerous tough adhesions were present at the base. In the upper lobe white concentric rings of apparently actinomycotic tissue were found. The lower part of the lobe was densely fibrotic.

The left lung weighed 226 grammes. Adherent pleurisy was present at the base. The substance of the lung was fibrotic in the lower part. There were two or three small white patches in its substance.

The small bowel and transverse colon were matted together with gelatinous adhesions. There was a moderate amount of free fluid in the abdominal cavity. Coming to the surface in the left inguinal region there was a large collection of pus. The abscess cavity was walled by a thick necrotic tissue and yellow gelatinous material. Other smaller abscesses existed scattered amongst adherent bowel.

The stomach was dilated with food. The peritoneal surface was gelatinous and infiltrated where it became a wall of a cavity. The mucous membrane was normal. The caecum was unaffected, on both peritoneal and mucosal aspects.

The liver weighed 849 grammes. It was very adherent to the right side of the diaphragm. The left lobe was unaffected. On the right lobe the peritoneum was very thick on the diaphragmatic surface. The right lobe of liver contained three or four patches of induration, taking the form of yellow rosetttes. The liver elsewhere was somewhat congested; otherwise it was normal.

The pancreas was indurated, but apparently not specifically affected.

The spleen weighed 56·6 grammes and was normal.

The right kidney weighed 113 grammes. Anteriorly the capsule was very thickened. In the upper pole both the capsule and substance were infiltrated with small patches of yellow tissue. Beneath and in contact with the capsule of the kidney was a large abscess in the retroperitoneal tissues, containing about 570 cubic centimetres (one pint) of thick, highly offensive pus. This abscess was found to originate in the bodies of the upper lumbar vertebræ.

The left kidney weighed 113 grammes. The capsule was thickened and somewhat adherent in places. On the anterior surface there was a large rosette of yellow necrotic tissue. The substance was somewhat fibrotic, but otherwise normal.

The bladder was normal.

The internal genital organs were normal, except that the peritoneal surface was affected.

The suprarenals contained minute yellow nodules.

The brain was soft and congested, but otherwise normal; no abnormality was detected in the cerebral membranes.

In the bones of the left elbow the periosteum was separated from the bone by serous fluid and the bone was ulcerated in patches with thick yellow pus.

The causes of death were (i) generalized actinomycosis and bone actinomycosis, (ii) toxæmia.

Histologically a ray fungus was demonstrated in lesions of lung, liver and kidney.

Commentary.

The duration of this case over a period of fifteen years with a period of apparent quiescence of eleven to twelve years between 1916 and 1927 are unusual features in actinomycosis, which, if visceral, as in this case, almost invariably progresses to a fatal issue.

A very interesting analysis of cases of actinomycosis recorded in America is contained in *Archives of Surgery*, December, 1925.(1) Dr. A. H. Sanford and Dr. Minna Voelker, of the Mayo Clinic, give a summary of 670 cases, the duration varying from one week to eighteen years. In none does there seem to have been such a long latent period as in the patient here recorded. In one instance a patient who, eighteen years previously, had had a tumour of the back removed by the local application of tincture of iodine, was admitted to hospital for actinomycosis in the same region and died of pulmonary involvement, but there appears to have been no definite evidence that the primary lesion was actinomycotic.

The *post mortem* appearances in the thorax in the case here recorded indicate that a more or less successful attack had been made on the disease in the lungs and parietal pleura. The distribution of the lesions would suggest that from the beginning there was a tendency to generalized infection.

The treatment in the later recrudescence of the disease was on the same lines as in the early stages from 1915 to 1916, but in spite of treatment by an autogenous vaccine and intensive iodine administration, the disease was steadily progressive. It was thought at times that the dosage of vaccine was excessive, so this was stopped for a while and new vaccines were tried at various times in different doses, but without effect.

With regard to the surgical treatment of the abscesses, free incisions were made and the infected areas were laid open over their whole extent, all actinomycotic tissue being curetted or cut away. Infection seemed to spread along fascial planes and in between muscles, making it very difficult to reach all parts of the infection. The raw areas were swabbed with tincture of iodine and the wounds were packed with gauze soaked in tincture of iodine, the gauze being removed after twenty-four hours. Although at times it appeared that the treatment had been successful, judging by the rapid healing of the operation areas and the improvement in general condition, after a few days or weeks swelling and fluctuation would recur in the same areas and rapidly develop into abscess formation.

Reference.

(1) A. H. Sanford and Minna Voelker: "Actinomycosis in the United States", *Archives of Surgery*, Volume XI, 1925, page 809.

Reviews.

STERILIZATION OF HUMAN BEINGS.

OF late years the presses have poured forth a flood of literature, by scientists and pseudo-scientists, on the menace of the socially inadequate to the future of civilization. In "Human Sterilization" Dr. J. H. Landman attempts to discover how far the jeremiads are justified in the case of the United States of America and how far efforts to prevent the multiplication of the unfit is likely to lessen their menace.[1]

In Part I of the book, entitled "Eugenics and Social Legislation", after giving an account of the history of human sterilization, the author states the views of the biological pessimists and contrasts with them those of the optimists who think that there is no cause for alarm. These are followed by details of the sixty-three laws dealing with human sterilization passed by twenty-seven of the United States. This section, together with Part II, "Human Sterilization and the Courts", is the most valuable part of the book. It describes fully the various legal pitfalls into which ill-considered legislation has plunged the authorities of various States and shows the means of avoiding them. Three leading appeal cases are related in considerable detail. This section should be invaluable to parliamentary draftsmen and members dealing with this matter.

Part III, "Biology and Human Sterilization", is less successful. Dr. Landman, who first became interested in the subject in 1927, seems to be a lawyer who has diligently studied his brief and industriously collected information about the scientific matter he proposes to handle. But he suffers the disability of a lack of professional training in his subject. This defect lessens the prestige that attaches to his statements, which are frequently erroneous and sometimes self-contradictory. We do not agree that the knowledge of human heredity is as slight as he insists that it is. Although he states that acquired characteristics are not inherited, he does not protest against the inclusion of syphilitics in the list of those justly sterilizable.

Part IV is devoted to the surgery of human sterilization, including the after-effects of the various operations. It suffers from the same weaknesses as Part III, but is probably adequate for its purpose.

In Part V the author asks and gives a reasonable answer to the question, "Whom shall we sterilize?" The procedure, while not being nearly such a panacea for social ills as its most enthusiastic advocates believe, has a definite but limited value. It must be reserved for those whose anti-social qualities are definitely transmissible, and it must not be used in cases of mere criminality, either for supposed eugenic reasons or, as apparently it has been used, as a punishment for crime.

Dr. Landman appears to have a low opinion of United States medical practitioners. He states, for example, that the liberal arts college graduate probably knows as much about heredity and psychiatry as the "M.D. physician"; that the operations to effect sterilization must be distinctly prescribed by law to prevent unscrupulous doctors from performing orchidectomy or oophorectomy instead of vasectomy or salpingectomy; that the desirability of sterilizing any particular person must be settled by judge and jury, because boards of doctors, besides being ignorant of their job, are liable to be swayed by political considerations. United States judges, not to mention juries, are doubtless devoid of such frailties. In spite of all this, however, we learn, with due humility, that the operation of tying the oviducts was introduced and perfected by a United States woman medical practitioner, who thereby achieved world fame. The author also thinks that vasectomy and salpingectomy should be performed only by genito-urinary specialists; by the same reasoning we presume that he would forbid any but gynæcologists to perform any operation upon a woman. The ignorance of simple Latin grammar which he shares with so many modern scientific authors, permits him to write of the *vas deferentes*. The exploits of the female of the United States species fill us with awe, since one bill was introduced to provide for the oophorectomy as well as vasectomy of those convicted of "murder, rape, highway robbery, chicken stealing, bombing, and theft of automobiles". The extensive bibliography is, as might be expected, almost exclusively United States publications. Many of the numerous statistics given are of very doubtful value. In spite of its defects, the book is a valuable review of its much debated subject.

[1] "Human Sterilization: The History of the Sexual Sterilization Movement", by J. H. Landman, Ph.D., J.D., J.S.D.; 1932. New York: The Macmillan Company. Demy 8vo., pp. 359.

THE DIGESTIVE TRACT AND ITS DISEASES.

"DISEASES AND DISORDERS OF THE DIGESTIVE ORGANS", by Dr. Adolphe Abrahams, is one of the series of "Pocket Monographs on Practical Medicine".[1] While miniature manuals enjoy a certain degree of popularity, it is not unlikely that their sale is supported by sluggish students and maintained by the mentally mediocre. Yet Dr. Abrahams's contribution to this series is worth reading. It contains a well proportioned summary of modern methods of diagnosis and of the principles of treatment. The various sections on the digestive tract and on the associated organs are viewed in their relation to one another and the symptom complexes originating elsewhere than in the diseased part are suggestively described. It is, of course, a book for the less well informed, and to them it may be cordially recommended.

[1] "Pocket Monographs on Practical Medicine: Diseases and Disorders of the Digestive Organs", by A. Abrahams, O.B.E., M.D., F.R.C.P.; 1932. London: John Bale, Sons and Danielsson, Limited. Foolscap 8vo., pp. 110.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 10, 1932.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

MENTAL HYGIENE.

THOUGH Juvenal recognized the excellence of "a sound mind in a sound body", it has taken us over two thousand years to return to the belief of Plato, "not that a good body will of its own excellence make the soul good, but on the contrary that a good soul will by its excellence render the body as perfect as it can be". The doctrine of mental hygiene, as we know it today, is built upon this belief.

In the fall of an apple lay the principles of gravitation; in the steam of a boiling kettle lay the dream of the locomotive; so in the committal of a lunatic there lay the idea of mental hygiene. Clifford W. Beers, who was certified insane in the year 1900, regained his sanity and reentered the world of his fellow men with a new vision. Knowing at first hand the treatment then meted out to the mentally afflicted, Beers was able to see the possibilities of a much needed reform; and with the zeal and courage of the true crusader he abandoned his business career and threw his entire energy into the cause of the mentally afflicted. In his remarkable book, "A Mind that Found Itself" (1908), he laid bare the story of his experiences in the mental hospitals; and twenty-four years ago, with the help of twelve other persons whose interest he had stimulated, he founded the first Society for Mental Hygiene.

The original aim of the mental hygiene movement was the alleviation of the sufferings and the better treatment of the mentally afflicted. Today its aims are very much wider and its importance is very plainly manifest. Mental hygienists not only seek to mitigate the suffering of those afflicted with the major psychoses, but to conserve the mental health of the whole community by preventing, as far as possible, all kinds of nervous and mental disorder or defect. They strive for the better care and training of the feeble-minded; they seek to exercise a guiding influence in the problems of education; they essay to deal more efficiently with delinquency; and in the wide realm of human behaviour they set out to help those who, by reason of their inability to make satisfactory adjustments to their environment, are rendered unhappy and socially inefficient.

Mental hygiene offers no panacea for the elimination of insanity or mental deficiency. Rather does it seek to prevent their production. It has no specific therapy to apply. Its fundamental aim is the enlightenment of the social conscience. In this it has become of world-wide significance. It is not the voice of some psychiatric prophet crying in the wilderness of modern medicine. It is a movement of almost national importance. At the first International Congress on Mental Hygiene, held in Washington on May 5 to 10, 1930, under the Presidency of Herbert Hoover, fifty-three different countries were represented, with a membership roll of over four thousand. The programme carried out at this congress covered all aspects of the problem of the maladjusted personality in relation to every department of human activity. A decade ago Professor Urwick, seeking a philosophy of social progress, found that society was "becoming clogged with its unfit, whom we insist upon keeping alive by checking every natural agent of selection". Mental hygienists now recognize the menace of mental ill health and instability which threaten civilization, and asserting their belief in social progress through scientific guidance, seek to prevent these evils and in the broadest sense to stem the tide of social subnormality, human wretchedness and inefficiency.

In Australia mental hygiene enthusiasts have not been idle. Victoria is taking the lead and some of the other States are slowly following. The Victorian Council for Mental Hygiene has issued its second annual report for the year ended June 30, 1932. The report is encouraging. Three subcommittees of the Council have been active—they are the Subcommittee on Education, Guidance and Prevention of Mental Ill-Health, the Subcommittee on Delinquency and Mental Deficiency, and the Subcommittee on the Early Treatment, Care and After-Care of the Mentally Afflicted. All three subcommittees have been trying to spread the doctrines of mental hygiene in such a way that nothing but good can result. Acting in conjunction with the Vocational Guidance Association of Victoria, the Council for Mental Hygiene has also undertaken the establishment of a Vocational and Child Guidance Centre. Dr. N. A. Albiston has been appointed Psychiatrist to the Centre, Mr. R. K. Whately, M.A., has been appointed Psychologist, and Miss C. P. Moffit, M.A., Social Worker. In this connexion the attention of readers is directed to an article by Dr. J. F. Williams, published in this journal on September 3, 1932. No one can deny that the Victorian Council for Mental Hygiene is working along right lines. Its members will look to medical practitioners for cooperation. Before cooperation must come understanding, and understanding will not be gained without effort. Medical practitioners owe it to their patients to make the effort.

Current Comment.

HEALTH AND THE WEATHER.

FROM time immemorial man has ascribed various influences, beneficent and malign, to meteorological phenomena. Man is able to observe the effects of the weather on his crops and his flocks and on the world about him; he sees the pools and streams dry up as a result of drought; he hears the sea birds cry as they fly to haven before the approaching storm; he sees the effects of the lightning in the splintered oak or the shattered gum tree; he feels the scorching of the tropical sun, shivers in the sleety blast of winter, and crouches in terror before the hurricane. Small wonder that he should associate changes in the weather with changes in his physical condition. Some interesting ideas on health as it may be affected by meteorological conditions are expressed by G. C. Simpson, a meteorologist, who discusses the matter from the point of view of the scientific layman.[1]

In his opening remarks, Simpson states that he deals "less with the ordinary opinions of medical men than with the medical opinions of ordinary men". He points out the fallacy of drawing conclusions from coincidences and from personal impressions; the opinion of a person who is guided by his impressions only is of no value, however long he has continued his observations. Simpson also stresses the danger of arguing from analogy, and instances the association of unpleasant odours with ill health, and of pleasant odours with good health; danger ensued when the carrying of flowers and perfumes became a method of guarding against infection.

Natural changes in barometric pressure are too slow to have any ill effects on their own account; but changes in pressure are accompanied by changes in the quantity of oxygen in the atmosphere; also low and high pressures are associated with cyclones and anticyclones with their characteristic weather conditions. It is the variations in the atmospheric constituents and the weather, not the alteration in barometric pressure *per se*, that influence human well-being.

Simpson discusses the importance of absolute humidity and relative humidity. When the absolute humidity is low, the quantity of water lost to the air by respiration is considerable. In very cold climates the loss of fluid by breathing is especially great; for cold air, even at saturation, contains only an insignificant quantity of fluid. When this fact is considered the reason for the great thirst experienced by polar explorers becomes obvious. The importance of the relative humidity (the ratio of the amount of water in the air to the amount that could be present at the same temperature) lies in the fact that on it depends the rate of evaporation from the skin. It is frequently stated that evaporation from the skin ceases when the air is saturated with moisture. Simpson disagrees with this. Providing the air temperature is lower than that of the skin, evaporation continues; the air is warmed when it comes in contact with the skin, and so is able to absorb more moisture. An example of this phenomenon is the "steaming" of a sweating horse on a humid day.

Simpson believes that of all effects of the weather on health and well-being, those of wind are by far the most important. Wind greatly increases the loss of heat from the body, provided the air temperature is lower than the body temperature. Even when the air temperature is higher, wind aids evaporation, and may have a tendency to cooling if the humidity is not too great; but it also has a tendency to heat the body. Normand has found that in calm air the human body can tolerate a temperature of 37·8° C. (100° F.) if the relative humidity is less than 90%, 48·9° C. (120° F.) if the relative humidity is less than 40%, and 60° C. (140° F.) if

[1] *Proceedings of the Royal Society of Medicine*, March, 1932.

it is less than 15%, but that death would occur, even if the air was quite dry, in a wind velocity of twenty miles per hour if the temperature was 53·3° C. (128° F.), or fifty-six miles per hour if the temperature was 47·2° C. (117° F.).

Simpson believes that the bracing quality of the air at health resorts is due mainly to wind and its cooling effect on the human body. The health-giving qualities of seaside resorts are often ascribed to ozone; but it has been found that the quantity of ozone in the atmosphere at such places is negligible and is probably less than in the polluted air of towns. The psychological effects of the sea odours are important.

The ætiology of sunstroke and the reasons for its peculiar geographical distribution have been the source of much argument. In concluding an instructive discussion on the subject, Simpson remarks that "if it is true that in parts of the world nearer the equator than India little or no sunstroke occurs . . . it will be found that in every case the air temperature is lower, or the air drier, or the wind velocity greater than it is in India when the sun attains the same altitude".

In the course of some interesting remarks on the chemical effects of radiation, Simpson states that Nature protects man from the more active chemical rays, by means of pigmentation. Why, he asks, is it considered good for people to obtain all the ultra-violet light possible, when Nature herself says it is not good for them? The administration of certain doses of ultra-violet light by a qualified medical practitioner is reasonable; there is no control over the dose obtained by sun-bathing or skiing without clothes. Simpson remarks: "One does not prescribe castor oil in that way." He believes that the effects of sunshine are mainly psychological. He quotes an experience of his own, remarking that after months of sunny weather at Simla a dull day gave him almost the same mental stimulus as that provided by a sunny day in England after weeks of dulness.

Simpson concludes with a discussion on the electrical field and the free electricity in the air. The first has no direct physiological effect, as it is unable to set up electrical currents in the body itself; the second is of no importance because it is negligible in amount, because the positive and negative ions tend to neutralize each other and because the electrical charge on the ion is lost in the damp, narrow passages of the respiratory tract. The electricity in the air during a thunderstorm, therefore, has no effect on the body. Thunderstorms have an indirect effect through the associated depressing weather conditions. It is not the thunderstorm that turns the milk sour, but the activity of organisms under hot and humid conditions suitable for their multiplication.

It is impracticable here to mention more than a few of the points made in Simpson's enlightening paper. The whole should be read by every medical practitioner able to obtain it. It is not often that the views of a non-medical scientist on a subject dealing with health find their way into medical literature; when they do and are of such value as these, they should be carefully studied. One of the most interesting features of the paper is the stress laid on the psychological effect of meteorological phenomena; perhaps this effect is unduly stressed; but there can be little doubt that it is the most important. An example is the refreshing influence of a Sydney "southerly" at the end of a hot day; the day's languor would not be so readily cast off if the influence were mainly physical.

ORGANIC AND INORGANIC IRON.

WITHIN recent years a good deal of stress has been placed on the value of certain impurities, notably copper and manganese, in medicinal preparations of iron. Experimental evidence goes to show that iron is of value as a builder of hæmoglobin only when copper is present in minute quantities as a catalyst. Obviously, the healthy animal obtains its supply of iron, and incidentally of copper, from its food. Presumably, a considerable quantity of such iron is in organic combination. The question then arises whether organic or inorganic iron is the more valuable. C. A. Elvehjem has recently conducted an experiment with a view to discovering the relative merits of organic and inorganic preparations of iron.[1] He remarks that the suggestion has been made that copper is necessary in the formation of hæmoglobin only when inorganic iron is given; he endeavoured to test the validity of this theory also. He points out in the first place that much of the iron contained in food is actually inorganic; for example, Hill has shown that the entire iron content of egg yolk is inorganic.

Elvehjem used white rats in his experiments, inducing anæmia by dietary measures. He employed hæmatin and ferric chloride as samples of organic and inorganic iron respectively. The administration of neither substance alone caused any increase in the formation of hæmoglobin. The addition of copper in each case caused a pronounced rise in the hæmoglobin content and a corresponding improvement in the animal's health. The most interesting feature, however, was the apparent superiority of the ferric chloride and copper over the hæmatin and copper. In no instance did the hæmoglobin content of an animal fed with hæmatin and copper rise above nine grammes per hundred cubic centimetres of blood; when ferric chloride and copper were given, the hæmoglobin content rose to thirteen or fourteen grammes per hundred cubic centimetres within two weeks. Elvehjem suggests that the organic iron has to be broken down to an inorganic salt before it can be absorbed.

The inference is that iron in its simplest form has the greatest therapeutic value. This is especially important from an economical point of view. The work also indicates the necessity for investigation into the organic and inorganic iron content of foods.

[1] *The Journal of the American Medical Association*, March 26, 1932.

Abstracts from Current Medical Literature.

SURGERY.

Post-Operative Obstruction.

D. GUTHRIE (*The Pennsylvania Medical Journal*, March, 1932) discusses the treatment of post-operative obstruction. He divides post-operative obstruction into two types, the dynamic or mechanical and the adynamic or non-mechanical. Either type may be caused by prolonged operations, by carelessly induced anæsthesia, by rough handling of tissues, by loss of heat and of body fluids, and by raw surfaces. For this reason the author does not use any form of self-retaining retractor, and he is opposed to rough dissection by gauze. Trauma to the upper portion of the small intestine is one of the commonest causes of post-operative ileus. Post-operative adynamic ileus is distinguished with difficulty from mechanical ileus situated low down or from early general peritonitis. A highly strung, nervous patient is restless; he has an anxious expression, a fast pulse, and a distended and silent abdomen; he also vomits. Treatment consists in the administration of large quantities of fluid with sodium chloride and glucose. This is given through a nasal catheter or by frequent gastric lavage through a Levine tube. The author recommends the method of Willard Bartlett as a means of differentiation between adynamic and dynamic ileus. A spinal anæsthetic is given, and if the bowels do not act owing to the resultant contractions of the small bowel, a simple enterostomy or a more radical operation is performed. Enterostomy may have to be performed for the more aggravated form of adynamic ileus. Post-operative intestinal obstruction is marked by recurring attacks of pain, by vomiting and by exaggerated peristaltic sounds audible on auscultation; intestinal patterns may be discovered on the abdomen. The sooner these patients are operated upon, the better. Quantities of fluid, isotonic and hypertonic solutions of sodium chloride, are given before operation, both by the subcutaneous and intravenous routes. Hypertonic solutions may stimulate peristalsis. Five to six litres of fluid are given during each period of twenty-four hours after operation, subcutaneously, intravenously or by the rectum. If the patient's condition is good and if no infection is present, the incision can be reopened and a mechanical blocking can be corrected; otherwise enterostomy should be performed. Frequently the cause of the obstruction disappears after enterostomy has been performed. A left rectus incision from the left costal margin is used and the peritoneum is opened sufficiently to permit removal of a single loop of bowel. A purse string suture is inserted into the loop between two clamps. The loop must be large enough to admit a small rectal tube, the end of which is inserted pointing down the bowel. The suture is tightened and its ends are tied around the tube. The procedure is repeated, the clamps are removed and the loop of bowel is returned to the abdomen. A skin silkworm suture is also tied around the tube. The author recommends constant attention to the tube, to keep it draining. He also suggests that irrigations should be carried out at least every hour with fluids injected without undue pressure.

Prevascular Femoral Hernia.

PREVASCULAR femoral hernia may have a congenital origin if an excessively long diverticulum of peritoneum accompanies the outgrowth of vessels into the limb bud. Most femoral herniæ traverse the saphenous opening, but those of the prevascular type continue beyond the opening in the femoral sheath and the sac comes to lie anterior to the vessels. Geoffrey Keynes (*The British Journal of Surgery*, July, 1932) reports a patient, aged twenty-three years, treated by removal of the sac after the femoral sheath was opened. The author employed the inguinal incision and used portion of anterior rectus sheath to fortify the reconstruction. The condition was bilateral, and the patient was free from recurrence for seven years after operation. Moschcowitz first described this condition *in vivo* in 1912. A somewhat similar condition, but considered to have been acquired, is reported in a man of sixty years, who, however, refused operative treatment.

Acute Perforated Peptic Ulcer.

J. GILMOUR AND J. H. SAINT (*The British Journal of Surgery*, July, 1932) have studied the late results of acute perforations of peptic ulcer. They review sixty-four cases occurring between the years 1922 and 1929. There was a sex incidence of ten males to each female. At the Royal Victoria Infirmary 90% of all perforated ulcers are situated in the duodenum. The histories of patients may be arranged in two groups, one extending over a few weeks and the majority lengthening into many years. The authors consider that the longer an ulcer has been present, the greater the likelihood of perforation. The treatment consisted of simple suture, the ulcer thereby being infolded. Unless there was much soiling and infection of viscera, the abdomen was closed without drainage. There were fifty-one cases in which less than twelve hours elapsed between perforation and operation, and in this group there was one death; among thirteen cases in which the interval was over twelve hours, there were two deaths. Of the patients under review, 36·4% required secondary operations for return of symptoms or complications. In twenty-seven cases of the series recurrences of symptoms occurred. Recurrence may occur up to five years. The authors outline the various methods employed in dealing with ulcer and state the arguments of the exponents of these methods. Duodenal stenosis following simple suture is unknown. Hæmorrhage and recurrence of perforation are exceptional sequelæ. Some Continental surgeons have reported extensive gastrectomy at the time of suture with a low mortality only, and by some this is ascribed to the use of splanchnic anæsthesia. In the discussion of the results of simple suture, the figures of other writers are given to show that complete absence of symptoms may be expected in from 25% to 60% of cases. The results of partial resection have been so satisfactory in the experience of Continental writers that the authors conclude it should be considered in preference to simple gastro-enterostomy when a secondary operation is planned. An extensive bibliography is appended.

The Earliest Surgical Treatise.

WARREN R. DAWSON (*The British Journal of Surgery*, July, 1932) gives an account of the Edwin Smith papyrus. Although it was discovered in 1862, very little was known of this papyrus until it came into the possession of the New York Historical Society in 1906. Professor Breasted has recently published a complete and reliable translation. It contains two distinct groups of writings. The first is a surgical hand book and differs from former discoveries in being arranged definitely as a book. It deals with injuries to the body from the crown of the head to the thorax; the portion dealing with the remainder of the body is missing. Forty-eight varieties of injury are described and the treatment is indicated. Many glosses are included at the end of certain descriptions. The Egyptians who lived thirty-five centuries ago knew that hemiplegia and paraplegia could follow head injuries. Some of the cases concerned injuries of the throat involving the gullet. A description includes priapism following cervical dislocation. Splints were used, composed of palm fibre or stiff linen rolls. Ointments contained honey as a basis, and often astringent or antiseptic herbs were added. The reverse of the papyrus is occupied with incantations to charm away the plague. It was commonly believed that plague was borne by winds, hence the restrictions on windows as recently as the great plague of 1665. Magic held supreme sway on the appearance of an illness which was not the direct result of a physical cause.

Cysts of the Thyreoglossal Duct.

F. W. RANKIN AND N. W. CRISP (*Western Journal of Surgery, Obstetrics and Gynecology*, March, 1932) epitomize the embryological development of the thyreoid gland and mention that failure of descent of the gland itself is one of the rarest congenital anomalies. The tract followed is usually posterior to the position later occupied by the hyoid bone. Complete failure of the duct to close is rare. Congenital tumours or

sinuses were reported by Sistrunk as being present in 31 of 86,000 consecutive patients at the Mayo Clinic. No age of life is exempt. Male patients predominate. The median swelling in the neck is usually noticed in early life, but apart from the tumour, little inconvenience results unless infection supervenes. Simple incision is the rule for drainage of pus. This is usually followed by formation of a sinus situated in the mid-line anywhere from the vicinity of the hyoid bone to the suprasternal notch. Very seldom is it necessary to inject opaque substances to outline the extent of the tract. The cyst or sinus is attacked through a transverse incision; it lies just beneath the raphe between the sterno-hyoid muscles. The central portion of the hyoid bone is removed and the tract cored from the deep muscles of the tongue right through to the *foramen caecum*. The remaining segments of the hyoid are approximated and a drain is used for the deeper layers for four to five days. The authors claim satisfactory results in 184 cases out of 200 in which treatment by this method was instituted.

Tumours of the Mesentery.

FRED W. RANKIN AND S. G. MAJOR (*Surgery, Gynecology and Obstetrics*, May, 1932) write about tumours of the mesentery. Tumours which originate within the leaves of the mesentery, because of their exceedingly uncommon occurrence, are looked on as unique surgical lesions. The authors review briefly twenty-two primary tumours of the mesentery. From an embryological point of view it is conceivable that mesenteric tumours may arise from displaced remnants of the genital gland, Wolffian body or its duct, or from the Müllerian duct. Nevertheless, there is no proof that any of the tumours described owed their pathogenesis to such embryonic remains. In the authors' opinion, their data contribute nothing to the origin of serous cysts, but because an epithelial lining was not demonstrable in these tumours, it is conceivable that they may have had their origin in hæmorrhage into the mesentery, the solid constituents of the blood having been absorbed. The endothelial lining of the chylous cysts favours the view that these neoplasms are due to dilatation of the lymph spaces rather than to effusion of chylous material into a preformed cyst. The sanguineous cysts would appear to be due to effusion of blood into the mesentery and do not seem to be hæmangiomatous. The pathogenesis of the lipomata and sarcomata is easier to understand than that of the cystic tumours. In this series the solid neoplasms occurred more frequently than the cystic variety, and sarcoma constituted the largest single group of the series. The tumours showed no definite predilection for either sex. The chylous cysts all occurred in the mesentery of the small intestine, whereas this was not the situation of the other types of cyst. The prognosis with the benign tumours is favourable, in spite of the pessimistic view expressed in the literature. In malignant tumour the outlook is unfavourable. The diagnosis of mesenteric neoplasms is difficult, but, given a mobile abdominal mass extrinsic to the gastro-intestinal tract, the possibility of mesenteric neoplasm should be borne in mind. The occurrence of these tumours is probably much more common than has been believed.

Abscesses of the Breast.

JOHN E. HOBBS (*Surgery, Gynecology and Obstetrics*, May, 1932) writes about a new method of treating breast abscesses. The same principles are maintained in treating breast abscesses as in treating suppuration elsewhere in the body, that is, incision and adequate drainage, rest, proper elimination, and symptomatic treatment. The most suitable time for incision is when definite fluctuation appears. Delay may mean extension and undermining of tissue with destruction of most of the lobules. In order to establish proper drainage by the usual method, it is necessary to make a large incision, and ofttimes counter-drainage is necessary. If it were possible to establish proper drainage with a small incision in the breast tissue, the ugly scarring could be avoided and normal breast tissue saved. De Lee states that in opening abscesses some respect should be paid to the appearance of the breast. Another important factor in treating breast abscesses is the question of dressings. If an abscess cavity is packed open with rubber tissue, gauze, or whatever seems suitable to use, the pack has to be renewed daily. This causes severe pain unless an anæsthetic is used, and surgeons do not like to anæsthetize patients for a change of dressings. On the other hand, if the patient is not narcotized, the cavity cannot be packed thoroughly. The third point for consideration is a satisfactory support for the breast, that is, one which will lift the breast and give moderate compression and maintain that support from one dressing until the next. The occurrence of breast abscesses can be prevented in great part by suitable prophylactic measures; but owing to neglect by the attending medical practitioner or by the patient, breast abscesses still occur frequently during the lactation period. It is important to establish proper drainage with minimum scarring of the breast. This can be done by making a stab wound large enough to accommodate a rubber tube 1·25 centimetres (half an inch) in diameter, which is made secure by suturing to the skin. Other drains and counter-drainage will be unnecessary. A Dakin tube is inserted through the drainage tube and the cavity is irrigated with hypochlorite of sodium, which is a disinfectant, destroyer of necrotic material, stimulator of granulation tissue, and a deodorant. It does not harm healthy tissue. Vaseline gauze can be removed and applied without pain to the patient. It protects the skin from the irritating and macerating effect of the discharge. It is imperative that this or some other oily dressing be used when the cavity is irrigated with hypochlorite of sodium. A gauze roll makes an excellent binder, for it gives support and compression, and serves as a protective dressing.

The Gall-Bladder.

I. F. RAVDIN AND C. G. JOHNSTON (*The Pennsylvania Medical Journal*, March, 1932) state that recent investigations into the anatomy of the biliary ducts and blood vessels revealed the text book arrangement in only 34·5% of cases. Abnormalities of blood vessels are numerous. Ligation of the hepatic artery causes death in twenty-four to forty-eight hours from hypoglycæmia. The gall-bladder of a dog has concentrated thirty-five cubic centimetres of bile to two cubic centimetres in seventeen hours. Its absorptive powers show selectivity. The normal gall-bladder absorbs no cholesterol or bile salts. The damaged gall-bladder secretes cholesterol. The introduction of pathogenic bacteria does not lead to disease unless the cystic duct is occluded, when empyema results. Emptying of the gall-bladder is the result of muscular action of its walls. Cholecystographic evidence of normal function makes it improbable that the gall-bladder is causing symptoms. Infection probably reaches it by the systemic circulation. In active inflammation the bile in the gall-bladder is partly replaced by an exudate rich in protein and calcium. Hence the occurrence of stones of uniform size or several groups following attacks of cholecystitis. Most stones are the result of infection, with the possible exception of the solitary cholesterol calculus, which may be of metabolic origin. No amount of duorenal drainage can cure chronic intramural infection. Later, biliary cirrhosis and pancreatitis may be added. Intravenous administration of glucose is a valuable help in overcoming the shock of operation. A high carbohydrate diet hastens the post-operative regeneration of liver cells. Morphine depletes the store of glycogen in the liver. Cholecystectomy is the operation of choice when the ducts are uninjured. Approximately 40% of patients who undergo cholecystostomy require a second operation within three years.

Thyreoidectomy and Drainage.

J. L. DE COURCY (*Archives of Surgery*, July, 1932) discusses the question of drainage after thyreoidectomy. He bases his remarks on the results obtained by him in twelve hundred operations. In all cases the author gave the patients iodine before operation. His usual operation consists of bilateral subtotal excision. He has found it necessary to use drainage in only 1% of cases. He concludes that drainage is usually unnecessary and may be harmful.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Ballarat on July 23, 1932. DR. A. C. H. SALTER, the President of the Ballarat Subdivision, occupied the chair; he was supported by Dr. B. M. Sutherland, the President of the Victorian Branch.

Renal Conditions and Renal Tests.

DR. G. T. JAMES read a paper entitled: "Bright's Disease: Its Classification" (see page 321).

DR. J. C. DOUGLAS read a paper entitled: "The Fundus in Nephritis" (see page 326).

DR. G. R. DAVIDSON read a paper entitled: "Renal Function Tests and Their Application to Surgery" (see page 329).

DR. RAYMOND GRIFFITHS read a paper entitled: "Some Aspects of the Renal Complications of Pregnancy" (see page 327).

DR. IVAN MAXWELL congratulated the readers of the papers on their excellent contributions. He said that he felt impelled to say a few words in defence of the biochemist and physiologist, although many practitioners, including some recent writers in THE MEDICAL JOURNAL OF AUSTRALIA, had criticized what they termed the "worship of the test tube"; nevertheless, all the clinical tests discussed by the readers of the papers that evening, were due in the first place to the work of biochemists rather than of clinicians. MacLean, whose urea concentration test and method for estimating blood urea were in common use, had been trained primarily as a biochemist. In future it seemed likely that more help would be rendered to the clinician by the biochemist than by the morphologist. There were many disturbances of function, the results of which could be demonstrated clinically, but which caused no structural change in the organs demonstrable either macroscopically or microscopically. In spite of the general conception that so-called nephrosis was a degeneration of the tubules alone, it was practically certain that the glomerulus was involved in this condition, as evidenced by gross albuminuria, and because it was found that when patients with nephrosis had been carefully examined and their histories investigated, some hæmaturia had occurred at one stage. As it was not considered that hæmaturia was a characteristic of pure primary nephrosis, many observers doubted the existence of primary nephrosis as a distinct entity, but rather as one form of chronic nephritis. Referring to the very great practical difficulties surrounding the question of renal function, Dr. Maxwell pointed out that there were at least five or six extrarenal factors concerned in the production of œdema, including blood pressure, the osmotic pressure of crystalloids and colloids in the capillaries and in the extracapillary tissues. One of the difficulties in reconciling the views of workers engaged in various fields of research was that the morphologist usually demanded to see some change in an organ before he could recognize change in function, whereas it was easily proved by the physiologist that albumin and globulin would appear in the urine from a kidney the renal vein of which was ligated for a few minutes and in which no demonstrable morphological change could be found.

Finally, Dr. Maxwell uttered a warning to clinicians not to put their trust in only one test for renal efficiency. He referred to a recent analysis by Dr. F. W. Green of a series of cases of prostatectomy, together with the record of the patients' renal function. Some patients had died who had had normal blood urea but subnormal urea concentration. In general, it was proved to be advisable to test renal function by at least two recognized methods before assuming that the function was within normal limits.

DR. W. I. HAYES expressed his appreciation of the papers and referred particularly to Dr. Griffiths's paper, which supported his own view, that the toxæmias of pregnancy should be treated more seriously than was often the case. The maternal mortality from renal complications was not diminishing in any part of the world, and possibly was even increasing. Renal complications occurred in about one-third of the deaths associated with child-birth. Dr. Hayes pointed out that it was usually not so much the severity of the toxæmia which determined the serious after-effects, but its duration. It was profitable to classify cases of eclampsia into those which occurred in young women—often very acute and frequently responding to adequate treatment—and those occurring in older women, usually *multiparæ*, in which chronic nephritis was so frequently present. At the Women's Hospital in Melbourne, in cases of preeclampsia it was the usual treatment in most cases to induce labour after a very short period of medical treatment. It was found that the renal function in albuminurics often deteriorated even during the period of apparently successful medical treatment. Cæsarean section had its use in those cases of severe eclampsia which did not respond to medical treatment within a short time.

DR. J. G. E. HAYDEN stated that he was glad to hear Dr. Maxwell's remarks and had reached the same opinion as he, that nephrosis was a very doubtful clinical entity. Nearly all the cases described did not exhibit at *post mortem* examination the pure characteristics of tubular degeneration, but almost invariably showed some evidence of glomerular damage. The clinical histories in most of the reported cases of nephrosis did not even conform to the author's definition. Van Slyke used hæmaturia as an absolute barrier, dividing the glomerulo-nephritic type from the nephritic and arteriosclerotic group. Dr. Hayden stated that if nephrosis did exist as a separate disease, it must be extremely rare.

In reference to the clinical classification of renal disease, he wished to thank Dr. James for a very thoughtful and reasoned paper, which must have required tremendous care in preparation.

There had been two tendencies in the nomenclature of renal disease, one was to multiply the subvarieties met with at *post mortem* examination, and the other endeavoured to reduce the clinical pictures to an impossibly simple form. Van Slyke's and McIntosh's use of hæmaturia as a differential symptom seemed to be useless, as many of his so-called nephrotic cases were characterized by hæmaturia, and it was well known that hæmaturia, even massive hæmaturia, occurred occasionally in arteriosclerotic renal disease.

In recent years many claims had been made for Addis's urea clearance test as being the most reliable of renal function tests. It was stated that renal impairment was directly related to the actual percentage number of glomeruli destroyed and that Addis's test could detect 40% destruction of glomeruli when the results of all other tests were normal. However, the result of the test was often below normal in pure arteriosclerotic disease, when the glomeruli appeared to be histologically normal, and it was suggested in these cases that the impairment was due to diminished renal blood flow. The test involved the estimation of the concentration of the urea in the blood and urine, and the urea clearance was defined as the number of cubic centimetres of blood per minute cleared of urea by renal excretion. Dr. Hayden felt that though this test was valuable, it must be assessed together with the clinical condition of the patient and the results of the other urinary findings.

DR. W. A. HAILES said that when the blood urea and the urea concentration tests were first evolved, it had been hoped that surgical prognosis would be easy; but it had been found that by no test could one predict how the bladder infection, which invariably followed the operation of cystotomy, would affect the already damaged renal tissues. Many such patients, who obviously had had good renal function as determined by tests, died within ten days of operation.

DR. W. T. GREENING said that six years before, in collaboration with Dr. Leo Doyle, he had investigated the kidney functions in a series of patients who were to be subjected to prostatectomy. It was found that the urea concentration test and the blood urea were normal in some

patients whose total non-protein nitrogen was much increased. In such cases two-stage prostatectomy was performed with consequent saving of life. Dr. Greening indicated that of the three tests, that for non-protein nitrogen was probably the most important.

DR. B. M. SUTHERLAND congratulated the speakers and said that he strongly supported the plea of Dr. Griffiths and Dr. Hayes for a complete investigation of urinary function in the early stages of pregnancy. A complete urine examination and a blood pressure estimation should be made when the patient was first examined, and these should be repeated at suitable intervals. In some cases, particularly in older women, the only indication of impaired renal function might be persistently low specific gravity of the urine, and this might precede a severe or fatal eclampsia in later pregnancy. He agreed with Dr. Hayes that Cæsarean section had an important place in the treatment of eclampsia, though it had not, as originally hoped, led to the saving of every patient and every child. It was necessary to observe closely all nephritic and eclamptic patients after their confinement for as long a period as practicable, because such patients not infrequently suffered permanent renal damage. The late Dr. Rothwell Adam had taught that any woman who developed so-called eclamptic fits after the age of forty, was probably already suffering from chronic nephritis. Dr. Sutherland's own experience had led him to believe that this was so.

Cholangitis.

DR. W. T. GREENING showed a child, aged five years, who gave a history of being restless and feverish and of not eating well. On examination hepatic enlargement was found. The Casoni test, the complement fixation test for hydatid disease, and the Wassermann test were performed, but no reaction was obtained. At surgical operation a section of the liver was removed for microscopical examination, and the pathologist reported the presence of "some fibrosis and round cell infiltration in the portal area".

DR. H. D. STEPHENS said that the enlargement of the liver seemed to be the only abnormal sign demonstrable on ordinary clinical examination. The fact that the mother had had four other healthy children and no miscarriages would seem to exclude syphilis, otherwise he would have suspected congenital syphilis as the probable cause of the illness. There was no record of any rise in temperature which might have indicated hepatic cirrhosis from a bowel infection. He had seen a somewhat similar patient, a boy of about ten years, referred to him by Dr. Derham; in that instance there had been an unexplained enlargement of the liver with upper abdominal pain. Tests for hydatid and X ray examination had revealed no abnormality. At operation a slightly distended gall-bladder was found, the fluid from which, on cultivation, yielded a streptococcus. The gall-bladder was drained and the boy made a slow but uneventful recovery. In the present case Dr. Stephens was at a loss to explain the underlying cause, but he would make a tentative diagnosis of hepatic cirrhosis of toxic origin and he would advise treatment to continue with big doses of iodides, as Dr. Greening was doing.

DR. S. O. COWEN said that he had seen a somewhat similar case in which the toxic cirrhosis had been due to the *Bacillus enterolyticus* of Gärtner. The condition was known as subacute proliferative cholangitis. Probably congenital syphilis could be excluded in the present case.

Hydronephrosis.

Dr. Greening also showed a patient who had suffered from attacks of renal colic on the right side. Two years before the patient's admission X ray examination after the first attack revealed no abnormality. After the second attack, a year later, a second X ray examination revealed no abnormality. After several further attacks a pyelogram revealed a much enlarged renal pelvis on the right side. Operation was performed on December 28, 1931. The ureter was dissected from the middle of the posterolateral aspect of the hydronephrotic renal pelvis, to which it was adherent, and a plastic operation on the ureteropelvic junction was performed by Finger's method. On July 19, 1932, intravenous pyelography revealed a normal renal pelvis on the left side, and on the right side the pelvis was half the size that it had been before operation.

Megalo-Ureter Without Obstruction.

DR. R. G. DAVIDSON showed a child, aged eight years, who had a history of enuresis and pyelitis of several years' duration. Urinary investigation and a pyelogram gave evidence of megalo-ureter without obstruction. *Bacillus coli* infection was present. The probable cause of the condition was, in Dr. Davidson's opinion, an absence of relaxation or achalasia of the uretero-vesical sphincter. The treatment adopted consisted of dilatation of the sphincter and renal lavage over a long period.

Lymphosarcoma.

DR. ARTHUR GUYMER showed a male patient, aged sixty-one years, who was admitted to hospital on June 14, 1932, complaining of having been "off colour" for the last nine months with severe attacks of colicky pain in the lower part of the abdomen. He had severe sweating at night, inability to do anything on account of increasing general weakness, and loss of appetite. He complained of constipation, getting gradually worse, and had lost about 6·3 kilograms (fourteen pounds) in weight. Also he had noticed a lump in each groin of four weeks' duration. He looked ill and anæmic. His heart and chest were normal.

His liver was enlarged to five fingers' breadth below the costal margin; it was smooth and soft to the touch. His spleen was not palpable. A vague mass was thought to be present in the left iliac fossa. The lymph glands along the iliac arteries and in the groins were enlarged to the size of walnuts; they were painless, soft, discrete, and could be moved about freely from one another. His leucocytes numbered 14,600 per cubic millimetre. A barium meal and barium enema revealed nothing abnormal.

During his stay in hospital he had an irregular temperature, rising occasionally to 38·9° C. (102° F.), whilst his pulse rate varied from 110 to 130 per minute.

Other superficial glands, submaxillary, cervical, axillary *et cetera*, rapidly became palpable, whilst the glands in the groin were becoming fixed to each other. Free abdominal fluid became evident and his general condition was deteriorating rapidly. A gland excised from the mass in the groin was examined by Professor MacCallum who reported as follows:

> The gland shows a fairly uniform structure in which the main constituent are cells whose character suggests an origin from the reticular elements of lymphoid tissue. There is a considerable variation in nuclear size and many scattered, large, and even "giant" forms are seen. They are in active multiplication and extension into the fat beyond the capsule occurs. The growth is one of the reticular variety of lymphosarcoma group. The myelocytes would appear to indicate severe damage to hæmopoietic tissue. Some of them may possibly be of the large lymphatic group which are often difficult to distinguish.

Dr. R. P. MCMEEKIN said he considered the condition to be a type of lymphosarcoma, as the glands in the groin were commencing to adhere one to another. The report of the microscopical examination by Professor MacCallum probably clinched the diagnosis. Deep X ray treatment might be of benefit, but this was usually only temporary when the temperature reaction was of the type shown in the present case.

Dr. S. O. COWEN thought that later in the progress of the disease the blood picture would probably approach that of a fully developed leuchæmia, and he would not be so pessimistic as Dr. McMeekin about the possible good effects of deep X ray therapy.

Congenital Dislocation of the Hip.

DR. W. SLOSS and DR. W. R. GRIFFITHS showed a patient illustrating the successful treatment of unilateral congenital dislocation of the hip by manipulation. The patient, who was aged two years and four months when treatment

was commenced, exhibited all the classical symptoms and signs of a dislocation of the left hip—marked limp on walking, with lurching to the left side, shortening of the limb, broadening of the perineum, telescoping of the limb, and displacement of the left trochanter above Nélaton's line. The diagnosis was confirmed by X ray examination.

Treatment was commenced on April 28, 1930. Under general anæsthesia the left adductor muscles were stretched and kneaded until full abduction could be obtained. The reduction was then effected by the method of Jones and Lovett, a snap being heard when reduction was effected.

The limb and pelvis were retained in a position of 70° of flexion and 70° of abduction without rotation. Unfortunately, the plaster was not successful in maintaining the reduction, and the process had to be repeated. This time the limb was flexed past a right angle and abducted to a position in which the knee was posterior to the plane connecting the anterior superior iliac spines, with the patella directed laterally. The plaster was applied in this position and pushed in posteriorly to hold the trochanter forward.

The plaster was replaced on June 23 and August 18, 1930. On October 27 and December 18, 1931, plaster was reapplied each time with a decreasing amount of flexion. In February, 1931, the plaster was removed and movement in a recumbent position encouraged. After six weeks walking was allowed.

In July, 1932, walking was possible without a noticeable limp, measurement showed that there were six millimetres (one-quarter of an inch) of shortening, and movement was free in all directions. There was still some muscular wasting. Dr. Sloss and Dr. Griffiths showed skiagrams illustrating the various stages in treatment.

DR. H. D. STEPHENS congratulated Dr. Sloss and Dr. Griffiths on their excellent result. Some workers had claimed 98% of cures in treating this condition, but nearly all authorities who had had much real experience of it, admitted that their results were not so good. A more general average was that one-third of the patients treated were cured, one-third improved, and in about one-third no improvement was achieved. In Putti's home district a large proportion of parents had themselves had congenital dislocation of the hip, and in consequence a large proportion of the children were born with it, and it was so common that it was customary to submit infants at birth to X ray examination as a routine measure.

Dr. Stephens cited the case of a baby who was brought to him for an opinion as to whether anything was wrong. Clinical examination revealed very little definite abnormality, and he had been tempted to tell the mother that the child was not abnormal. He had noticed, however, that the mother herself came into the room with a limp, and he asked her whether she had had congenital dislocation of the hip, and, on being told that she had, the baby was examined radiologically and it was found that there was a mild degree of congenital dislocation also present in the child. In the early days of his practice he had recommended postponement of manipulation and plastering in these cases until the child achieved control of its urine. He had found, however, that children suffering from dislocation frequently were late in gaining this control, and he now followed Putti's advice and put the child up with an abduction pad early in infancy. The treatment was a great trial to the parents, as it was almost impossible to keep the child clean, the plaster becoming soiled and softened with urine *et cetera*. In many cases of congenital hip disease treated in this way the X ray appearances later in life resembled those characteristic of Perthes's disease.

Friedman's Test for Pregnancy.

DR. W. R. GRIFFITHS showed the ovaries from an adult virgin female rabbit which had received injections of urine from a woman with six weeks' amenorrhœa. Owing to technical difficulties, the injections were not made intravenously, as recommended by Friedman, but intraperitoneally. On the first day two injections of five cubic centimetres and eight cubic centimetres respectively were given, and on the second day two injections of ten cubic centimetres each. The animal was killed on the morning of the third day, and the ovaries showed numerous *corpora hæmorrhagica*. The result of the test was presumably positive.

PROFESSOR R. MARSHALL ALLAN said that he considered Friedman's test was of extreme value, but the veins in the ears of small animals were difficult to inject, and intraperitoneal injections sometimes killed such animals by the toxicity of the urine, especially in the presence of bacilluria. In favourable cases, however, the accuracy of the test was about 98%.

DR. A. E. TAYLOR said that he had been interested in Friedman's test, and in the absence of the supply of rabbits he had attempted to perform a test by the intraperitoneal route on frogs, but found that these animals invariably died.

DR. H. PEARCE presented a young man suffering from a linear nævus extending across the right pectoral region, axilla, and down the anterior aspect of the upper arm.

Bilateral Tremor.

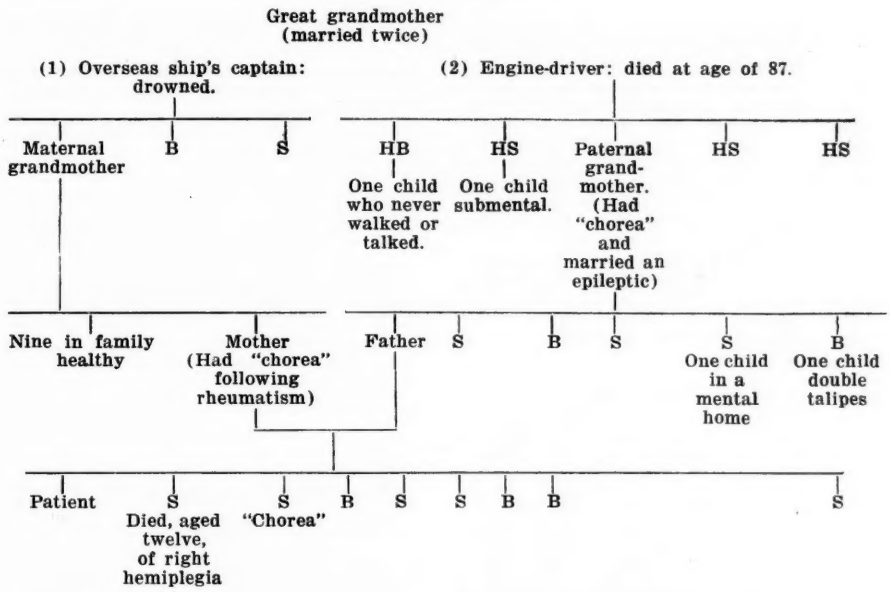
DR. G. T. JAMES presented a patient suffering from bilateral tremor. The patient was seventeen years of age and complained of difficulty of speech, of difficulty in walking, and of tremors in both hands and arms. She had been troubled with "growing pains" earlier in life, and had had sore throats. Her tonsils were removed eighteen months ago. Up to the age of twelve she was quite normal mentally and physically. At this time she had a fall downstairs, which apparently caused no injury. About the same time she had an attack of measles. She was quite normal physically and mentally until after the age of twelve years. Her mother noticed the first indication of anything abnormal, namely, a certain clumsiness in walking. Six months later the school teacher noticed that the patient was constantly "shivering", the shivering being confined to the head and neck. Later, the shivering became a definite tremor of hands, mostly the right. This was so pronounced that the child began to write with the left hand. Lately writing had become almost impossible and walking increasingly difficult.

The family history is shown in the accompanying genealogical tree.

Examination of the nervous system disclosed: (i) no sign of any involvement of the pyramidal tract; (ii) no abnormality of sensation; (iii) cranial nerves—sluggish response to accommodation, no nystagmus, normal eye movements, normal fundi; (iv) normal response to cerebellar tests; (v) a definite rigidity of the lower extremities, a dysarthria, and coarse tremors of both arms and hands; (vi) no response to the Wassermann test, slightly increased pressure of the cerebro-spinal fluid, which was clear, had a normal cell content, normal chlorides, and which contained no globulin.

Dr. James said that the case was referred for diagnosis, and discussion on the influence of the ætiological factors, hereditary and otherwise.

DR. R. P. MCMEEKIN referred to the bad family history and the tremor of basal ganglion type as in Parkinsonism. He pointed out that rigidity of the arms was not marked, nor was the mask-like facial expression well developed. There was lessened frequency of blinking and there was slowing of accommodation contraction, especially in the left eye, and some evidence of mental deterioration. Dr. McMeekin considered that the signs must be explained by several lesions; the condition, he thought, might be an abiotrophy of congenital origin or an inflammatory condition associated with the measles. He thought the outlook was very bad. The rigidity had been found to respond not infrequently to large doses of hyoscine, belladonna and stramonium. The gait and speech might be helped by these, but it was necessary to use the drugs in doses considerably larger than those stated in the pharmacopœia. It might be necessary to give up to 200 minims a day of the tincture of stramonium. It had been recently reported from America that good results were obtained from giving atropine by mouth in 1% solution, of which twenty minims three times a day were given with benefit.



Mediastinal Tumour.

DR. C. RICHARDSON showed a male patient, aged four years, who had collapsed under a general anæsthetic while being circumcized at the age of ten days. The patient was then treated for "fortorospasm". At the age of six months the child developed persistent croup and signs of a mediastinal tumour were manifest. The diagnosis was confirmed by X ray examination. At the age of ten months the child was given deep X ray therapy and definite relief from obstructive symptoms was obtained. At the age of twelve months a further application of deep X ray therapy was made, followed by definite relief. At the age of two years and three months a third dose of deep X ray therapy was given. Dr. Richardson pointed out that since the last application of X rays the child had had no further recurrence of symptoms and had survived both measles and whooping cough.

DR. H. L. STOKES said that in the absence of any X ray evidence, he would have diagnosed, from the history and clinical examination, a condition of persistent thymus or an enlarged heart. The fact that the apparent tumour had not decreased in size in response to deep X ray therapy tended to exclude the condition of enlarged thymus.

DR. R. P. MCMEEKIN said that in adults mediastinal tumours of the lymphosarcoma and Hodgkins type, causing symptoms similar to those of Dr. Richardson's patient, frequently responded to deep X ray therapy, the symptoms subsiding and the tumour decreasing in size. In the present case he doubted whether the improvement had been due to the deep X ray therapy.

DR. JOHN HAYDEN said that he was inclined to the view that the enlargement was due to a persistent thymus or an enlarged heart, probably the latter.

Solitary Hepatic Abscess Following Acute Appendicitis.

DR. N. A. LONGDON showed a boy, fifteen years of age. While in hospital for a linear fracture of the cranial vault, the patient developed acute appendicitis, on the twenty-eighth day. At operation a perforated gangrenous appendix was removed along with some enwrapping omentum. Purulent fluid was found in the pelvis. The pelvis and the right paracolic gutter were drained with split rubber tubes through appropriate stab incisions. The patient was given eighteen cubic centimetres of gangrene antiserum intramuscularly, and two cubic centimetres of a 2% solution of acriflavine intravenously. He was treated in the full Fowler position and was given rectal injections of glucose and saline solution every four hours and hypodermic injections of morphine as required for restlessness or pain.

Drainage from the tubes was free, vomiting ceased after forty-eight hours, and the moderate distension disappeared when the bowels were opened with an enema on the fifth day. The temperature, however, remained elevated to about 38·3° C. (101° F.) and the pulse rate to about 120. This persisted for three weeks, and the patient's only complaint was of a constant dull pain in the right loin. Repeated urine and rectal examinations revealed no abnormality, the white blood cell count was 14,000 per cubic millimetre, and the wounds had practically healed.

Radiological examination did not reveal any elevation or irregularity in outline of the diaphragm nor any definite opacity of the costo-phrenic angle. The liver edge, however, was palpable one finger breadth below the right costal margin.

On the twenty-fourth day after operation the patient had a rigor, looked very "toxic", and the liver edge was more readily palpable. An exploring needle was introduced through the ninth right intercostal space, near the post-axillary line, and foul-smelling colon bacillus pus was located at a depth of about 8·75 centimetres (three and a half inches). Portion of the ninth rib was resected under general anæsthesia, and a needle introduced into the abscess cavity was seen passing through the diaphragm. With the needle *in situ* the usual trans-thoracic approach was used to drain the abscess, which was found to be a solitary one containing about 90 cubic centimetres (three ounces) of pus, and situated at a depth of nearly 2·5 centimetres (one inch) from the superior surface of the right lobe of the liver. It was dealt with in the usual manner, except that accidental contamination of the pleural cavity occurred, but recovery was uneventful.

Dr. James enumerated the somewhat unusual features of the case as follows:

1. The occurrence of a solitary abscess in the liver from an infected embolism carried by the portal vein.

2. The association of such a definite abscess with a white blood cell count of only 14,000 per cubic millimetre.

3. The absence of more definite radiological evidence, probably explained by the depth of the abscess within the liver.

4. The constant localization of the pain to the right loin.
5. The good fortune or acumen, or both, of Dr. Wilson in striking the pus with the first exploratory puncture.

DR. ALAN HAILES quoted a case at the Melbourne Hospital in which dulness to percussion extended up to the lower angle of the scapula on the right side, but no pus was obtained on acupuncture. The patient recovered, but the dulness still persisted and the diaphragm was still shown by X ray examination to be elevated. As a result of this and similar cases Dr. Hailes said he had lost faith in the demonstration by X rays of elevation of the diaphragm as evidence of subphrenic abscess.

DR. R. P. MCMEEKIN said that it was not very uncommon to see paralysis of one side of the diaphragm following pneumonia. This led to an X ray appearance resembling that described by Dr. Hailes, and was probably due to involvement of the phrenic nerve in the pleurisy complicating pneumonia.

Splenomegaly of the Banti Type.

Dr. Longdon also showed a male patient, aged twenty-eight years, who was admitted to hospital in November, 1927, following two attacks of hæmatemesis. He looked anæmic and his spleen was palpable four fingers' breadth below the costal margin, and ascites was present. The Wassermann test yielded no reaction. Blood examination revealed 4,500,000 red cells per cubic millimetre, 2,500 leucocytes per cubic millimetre, with a normal film. *Paracentesis abdominis* yielded twelve pints of fluid. The condition was diagnosed as Banti's disease, and splenectomy was performed in January, 1928.

The spleen, which was densely adherent and fibrotic, measured 22·5 by 12·5 centimetres (nine by five inches) and weighed 707 grammes (one pound nine ounces), and blood transfusion was necessary after the operation. After a stormy period, in which the wound broke down and in which bilateral pleural effusions developed, the patient recovered.

Dr. Longdon said that the patient had remained fairly well since, except for an attack of quinzy in February, 1930, and a hæmatemesis in September, 1931. There had been an apparent increase in the size of the liver, which, according to the hospital records, was one finger breadth below the costal margin in May, 1928, two fingers' breadth in February, 1929, and three fingers' breadth in September, 1931. Periodical blood counts during the last four years showed that the red count had fluctuated between three and a half and four and a half millions, while the white count has gradually increased from two and a half thousand to seven thousand. At the time of the meeting the red cells numbered 6,000,000 per cubic millimetre and the white cells 7,000.

Dr. Longdon pointed out that at the time of operation the condition was regarded as Banti's disease, which, owing to the presence of ascites and a liver which was not above normal in size, was considered to have entered the third stage, and in which, therefore, prognosis was not good. The subsequent course of the case suggested that many of the patients with Banti's disease who were apparently entering the terminal stages with ascites, might derive much temporary benefit from splenectomy. It also emphasized the fact that the ascites was not always due to a contracting cirrhotic liver.

DR. R. P. MCMEEKIN said that he considered the prognosis that of progressive cirrhosis of the liver, which invariably followed splenectomy in these cases.

DR. W. T. GREENING said that ascites should not be a contraindication to operation. Early ascites was probably due to a perisplenitis and not to obstruction. This theory would also explain why ascites usually cleared up after splenectomy.

DR. A. W. HAILES said that at the Mayo Clinic it was considered that one-sixth of the blood in the portal circulation was contained in the splenic vein, and in Banti's disease the huge enlargement of the spleen caused the splenic circulation to represent much more than one-sixth of the total portal circulation, and ascites therefore resulted from the consequent obstruction. Splenectomy relieved this load, leading to disappearance of the ascites.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Bayldon, Francis Wood, M.B., B.S., 1930 (Univ. Sydney), Taree.

The undermentioned have been appointed members of the Victorian Branch of the British Medical Association:

Buxton, Thomas Fowell, M.B., B.S., 1931 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

Harry, Norman Marshall, M.B., B.S., 1931 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

Downes, Henry Erskine, M.B., B.S., 1926 (Univ. Melbourne), D.P.H. (Sydney), c.o. Commonwealth Department of Health, Chancery House, Bourke Street, Melbourne, C.1.

Australasian Medical Publishing Company, Limited.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE AUSTRALASIAN MEDICAL PUBLISHING COMPANY, LIMITED, was held at The Printing House, Seamer Street, Glebe, on August 31, 1932, DR. T. W. LIPSCOMB, the Chairman, in the chair.

Directors' Report.

The Directors' report was presented as follows:

The Directors submit their report for the past year and the balance sheet as at June 30, 1932, together with the profit and loss account for the twelve months ended June 30, 1932.

The Company suffered a severe loss when Dr. R. H. Todd died on December 14, 1931, shortly after his return from a visit abroad. Dr. Todd was Secretary of the Company and had been actively connected with it since its inception. Mr. J. H. Noldt, who had been Acting Secretary during his absence, was elected Secretary.

The work of THE MEDICAL JOURNAL OF AUSTRALIA and of The Printing House, under the capable control of Dr. Mervyn Archdall in the Editorial Department, and Mr. A. Simpson in the Printing and Publishing Department, continues to meet with success. THE MEDICAL JOURNAL OF AUSTRALIA has retained its popularity and position as a foremost scientific publication; and a large amount of new business has been obtained in the Printing Department, although this has been offset to a considerable degree by the reduction in prices.

Despite this reduction, caused mainly by the economic and financial position of the Commonwealth, the result of the transactions of the Company for the twelve months under review has been a small surplus.

As a result of the year's work the Directors have been enabled to authorize payment of a portion of the accrued debenture interest.

Following the passing of the *New South Wales Interest Reduction Act*, 1931, interest on series "A" debentures was reduced from 5% *per annum* to 4% *per annum*, and on series "B", "C" and "C2" from 10% *per annum* to 7½% *per annum*, the reduction to take effect as from October 1, 1931.

A refund of unemployment relief tax which had been deducted from interest paid in October, 1931, to non-resident debenture holders whose income from New South Wales did not exceed £100 for the year ended June 30, 1931, was obtained from the Commissioner of Taxation and has been credited to the individual debenture holders' accounts. This refund will be added to the amount of interest to be paid within the next few weeks.

Sir Henry Newland and Dr. D. G. Croll retire from office by rotation, in accordance with the Articles of Association (Article 39). They are eligible and present themselves for reelection.

T. W. LIPSCOMB,
Chairman.

Election of Directors.

Sir Henry Newland and Dr. D. G. Croll were reelected to the Board of Directors.

Post-Graduate Work.

DEMONSTRATIONS IN MELBOURNE.

THE Saint Vincent's Hospital (Melbourne) Post-Graduate Committee has arranged to hold a series of practical post-graduate demonstrations in the out-patient department of Saint Vincent's Hospital at 4.30 p.m. on Tuesday and Friday afternoons, commencing on September 13 and ending on October 14, 1932. The syllabus of the demonstrations is as follows.

September 13, 16: "Technique of Local Anæsthesia", Dr. Leo Doyle.

September 20, 23: "The Diagnosis of Gastric Disorders", Dr. H. B. Devine.

September 27, 30: "Practical Details in the Treatment of Fractures by Bohler's Method", Dr. T. King.

October 4, 7, 11, 14: "Psychiatric Principles in General Practice", Dr. John F. Williams.

Research.

THE WILLIAM GIBSON RESEARCH SCHOLARSHIP.

THE Council of the Royal Society of Medicine has awarded the William Gibson Scholarship for Medical Women to Dr. Audrey E. Russell, of the Institute of Anatomy, University College, Gower Street, London. Dr. Russell proposes to carry out a research on the physiology of lactation and the factors promoting successful lactation in women.

Obituary.

THOMAS WALKER SINCLAIR.

DR. THOMAS WALKER SINCLAIR, whose death occurred at sea on January 30, 1932, was born in Melbourne in 1876. He was the son of the late William Sinclair, who was for many years on the staff of the Melbourne City Council. He graduated in Medicine at the University of Melbourne in 1897 with first class honours. He won the Beaney Scholarship in Operative Surgery and the Beaney Scholarship in Bacteriology and Pathology. In 1901 he obtained the higher degree of Doctor of Medicine. In 1903 he took the Diploma in Public Health of the Royal College of Physicians (London).

Sinclair held many appointments. He was Resident Medical Officer at the Melbourne Hospital in 1898. He was Assistant Demonstrator in Pathology and Bacteriology at the University of Melbourne in 1899 and 1900, and Honorary Pathologist to the Eye and Ear Hospital in 1900 and 1901. He went to England and became Assistant Resident Medical Officer of the Metropolitan Asylums Board, London. He spent twelve months at the Eastern Fever Hospital, Homerton, and several months at the Gore Farm Small Pox Hospital, Dartford. He returned to Australia and was appointed Honorary Assistant Demonstrator in Anatomy at the University of Melbourne in 1904. In the following year he went to Sydney as Assistant Medical Officer of Health to the Metropolitan District. He held this post until 1913. During his residence in Sydney he lectured on sanitary law, infectious disease, food inspection and hygiene at the Technical College, Sydney. For several short periods he acted as City Health Officer at Sydney.

In 1913 Sinclair was appointed Medical Officer of Health to the Melbourne City Council. He was the second full-time medical officer to hold this appointment. During his term of office he earned the respect and gratitude of the City Councillors. When he took up his duties he reorganized his department and divided the city into health districts, which were assigned to health inspectors. He took an active interest in child welfare work, and with Dr. W. Kent Hughes represented the City Council on the Executive Committee of the Victorian Baby Health Centres Association and the City Centres Committee. It was during his term of office with the Melbourne City Council that the first child welfare centre building was erected in Abbotsford Street, North Melbourne; and much useful work was carried out under his supervision. He acted as Chairman of the Committee controlling the campaign against poliomyelitis and directed the special campaign against diphtheria, inaugurated by the Health Committee of the Council in 1924. In 1924 he was granted eight months' leave of absence to visit Europe and America in order to obtain first hand information regarding recent developments in public health work and administration. He also acted as Lecturer on Public Health at the University of Melbourne.

Sinclair's work for the Melbourne City Council was characterized by efficiency, thoroughness and integrity. When he resigned in 1927, owing to ill health, the City Councillors were unanimous in their appreciation of his services.

Dr. John Dale writes:

Dr. Sinclair was my predecessor Medical Officer of Health of the City of Melbourne, and it was with the greatest regret that I and the whole staff of the Department heard of his death.

Dr. Sinclair's reputation as a scholarly and efficient health officer was known to me in Western Australia and was more than confirmed by my experiences in taking over his work.

All those modern features of the departmental work of which the city may well be proud, namely, preventive work against diphtheria and other infectious diseases, the campaign against poliomyelitis, and the child welfare work, were all inaugurated during Dr. Sinclair's tenure of office.

Dr. Sinclair's keen interest in his work was maintained after his resignation, and his periodical visits will be greatly missed.

PETER BRUCE BENNIE.

WE regret to announce the death of Dr. Peter Bruce Bennie, which occurred on August 25, 1932, at Sandringham, Victoria.

Correspondence.

THE BITE OF LATRODECTUS HASSELTII.

SIR: With reference to the "katipo" (*Latrodectus hasseltii*), I wish to state that that spider is commonly found in the Philippines, especially in the southern islands of the archipelago, where it is called "aloy" by the natives.

This last year four severe cases of "katipo" bites were brought to the Mission Hospital from among the labourers of a pineapple plantation. They presented the classical symptoms described by English physicians in Australia—profuse perspiration, elevated blood pressure, rapid and laboured respiration, slow pulse, numbness, weakness, and paralysis of the lower limbs preventing patients from

standing or walking. These were not all the symptoms, however, for there were others like vomiting, chills, dizziness *et cetera*, but those enumerated above were the ones found in all the patients. All these patients had identified the spider and had described it as a small, black spider with a red back. The spider has been identified at the United States National Museum at Washington, D.C., as the *Latrodectus hasseltii*.

Two other patients, pineapple labourers, had also been bitten by the "katipo" this last year, but had not developed symptoms severe enough to make them come to the hospital.

Our only treatment in the four cases that were admitted and had alarming symptoms, was 25% solution of magnesium sulphate administered intravenously—10 cubic centimetres to the dose and repeated as necessary. Two to four doses were sufficient. We believe this treatment is very efficacious and we do not get over anxious over our patients bitten by the "katipo".

As cases of bites of the "katipo" are very common in Australia and New Zealand, I would like to suggest that magnesium sulphate be tried in these cases. I have always used the Parke, Davis and Company's preparation which comes in ampoules, but other makes may be just as good.

Yours etc.,

CESAREO DE ASIS, M.D.

Mission Hospital,
American Board of Missions,
Cagayan, Oriental Misamis,
Philippine Islands,
June 30, 1932.

Books Received.

ORTHOPÆDIC SURGERY, by Walter Mercer, M.B., Ch.B., F.R.C.S., F.R.S., with a Foreword by John Fraser, M.C., M.D., Ch.M., F.R.C.S.E.; 1932. London: Edward Arnold and Company. Medium 8vo., pp. 678, with illustrations. Price: 32s. 6d. net.

RECENT ADVANCES IN OBSTETRICS AND GYNÆCOLOGY, by Aleck Bourne, M.B., F.R.C.S., and Leslie Williams, M.D., F.R.C.S.; Third Edition; 1932. London: J. & A. Churchill. Demy 8vo., pp. 413, with 87 illustrations. Price: 12s. 6d. net.

Diary for the Month.

SEPT. 13.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 20.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 21.—Western Australian Branch, B.M.A.: Branch.
SEPT. 23.—Queensland Branch, B.M.A.: Council.
SEPT. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 28.—Victorian Branch, B.M.A.: Council.
SEPT. 29.—South Australian Branch, B.M.A.: Branch.
SEPT. 29.—New South Wales Branch, B.M.A.: Branch.
OCT. 4.—New South Wales Branch, B.M.A.: Council.
OCT. 5.—Victorian Branch, B.M.A.: Branch.
OCT. 5.—Western Australian Branch, B.M.A.: Council.
OCT. 6.—South Australian Branch, B.M.A.: Council.
OCT. 7.—Queensland Branch, B.M.A.: Branch.
OCT. 7.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates of Local Associations.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes*, sought, etc., see "Advertiser," page xiv.

ADELAIDE CHILDREN'S HOSPITAL (INCORPORATED), ADELAIDE, SOUTH AUSTRALIA: Medical Superintendent.

AUSTIN HOSPITAL FOR CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Radiologist.

BALONNE HOSPITAL BOARD, QUEENSLAND: Medical Officer.

DEVON PUBLIC HOSPITAL, LATROBE, TASMANIA: Surgeon Superintendent.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Seven Resident Medical Officers.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.